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A PLEA FOR THE NEUROTIC THEORY OF GOUT.

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"The difficulties and refinements relating to the disease itself . . . . I will leave for Time, the guide to truth, to clear up and explain."
A Treatise on Gout and Dropsy, Sydenham, 1683.

I VENTURE in the present essay to put forward some arguments which tend, in my opinion, to sustain the thesis, that gout is a malady of neurotic origin.

In the first place, I wish to clear myself from a charge that may be brought against any one who seeks to promulgate such a view. I must confess that I am not bound to any one-sided theory of disease, in general; that I am not a rampant neuropathologist; and that I am constantly endeavouring to seek exactness and truth in medicine. And, having said this much, I may pass on to state that, having been a pupil and intimate friend, for many years, of the late Professor Laycock, I have not failed to imbibe, and to weigh very carefully, the doctrines laid down by him on many matters of profound interest in practical medicine and pathology. I have carried these views along with me, and have applied them, day by day, to the facts which have come before me in the course of hospital experience. At my hands, therefore, these views have received the roughest and most work-a-day application.

"Gout is primarily and pre-eminently a neurosis." So wrote Laycock to me a year before his death. His great predecessor in the Edinburgh Chair of Practice of Physic, Cullen, taught the same doctrine.

"Gout is manifestly an affection of the nervous system, in which the primary moving powers of the whole system are lodged. The occasional or existing causes are almost all such as act directly upon the nerves or nervous system, and the greater part of the symptoms of the atonic or retrocedent gout are manifestly affections of the same system. This leads us to seek for an explanation of the whole of the disease in the laws of the nervous system, and particularly in the changes which may happen in the balance of its several parts. Of the several pyrexiæ which are diseases of the sanguiferous system, some are with, and others without a considerable affection of the nervous system: pyrexia and neuroses, therefore, are necessarily and unavoidably mixed more or less with one another. Of those which are mixed, gout is a principal instance, in so far as it is an inflammatory disease; like rheumatism, it is placed among the pyrexiæ, but it is among the limits between pyrexia and neuroses, and shows more than any other pyrexia does of an affection of the nervous system."[1]

Cullen states that he adopted his views as to the nervous causes of gout from Stahl.[2]

Within the last few years, large progress has been made in the study of nervous disease, and quite recently, in particular, has light been shed upon various affections of the joints, which can now with certainty be relegated to disorders of the central nervous system.

Few writers have approached this subject since Professor Laycock put forward his views, and these, indeed, were never fully developed by him.

His most minute description is given as follows: "Excessive activity of the nervous system, or of portions of it, becomes a highly predisposing cause of the neuroses, as of

[1] 'First Lines of the Practice of Physic,' vol. ii. part i. chap. xiv. Edited by John Thomson, M.D. Edin., 1827.

[2] 'Theoria Medica Vera,' &c. G. E. Stahl. (Halle, 1737.) "De Doloribus Spasticis Arthritico-Podagricis," § xxxviii. p. 1040.

other general diseases. . . . These excesses, when habitual or long continued, are apt to develop hereditary tendencies. Thus, great mental labour, drunkenness, strain on special nerves, and the like, of parents, are often manifested in children as neuroses. Here the influence of the nerve-centres on the nutrient forces of tissue is shewn, as in hereditary insanity, epilepsy, hysteria, angina pectoris, *and even gouty diseases in general*, which, primarily, are neurose degenerations in the nutrition and transformation of certain tissues."[1]

The exceptions I have alluded to, I must pause to refer to briefly. The first consists in a paper which was written by Dr. Austin Meldon of Dublin,[2] in which he makes objection to the uric acid theory as explaining fully the cause of gout. He quotes cases to show, as is well-known and recognised, that uric acid and urates may exist in the blood to large extent without giving rise to gout, or being the result of gouty taint or inheritance, and he invokes the nervous portion of Cullen's theory to "complete the chain." His theory is as follows. He believes that the presence in the blood of uric acid and of soda, in some form, constitutes the predisposing cause of gout. "Nerve-force, when in a healthy condition, preserves these two in a fluid state, separately, in a condition in which they may be eliminated by the skin, kidneys, or bowels. As soon, however, as this nerve-influence is lessened, these two substances unite in the tissues most removed from the brain and centre of circulation. Irritation and inflammation excite the nervous system to increased energy, and the disease, for the time, is arrested." Dr. Meldon lays stress upon the fact of the attacks occurring mostly at night, when the nerve-force and circulation are feeblest, upon the common implication of the big toe, and notes the marked effect of depression of the nervous power as an important factor in their production. This theory, then, is a compound or humoro-neurotic one.

The second exception I have referred to above is a very notable one. I allude to the views of Dr. Edward Liveing which were published in his masterly exposition of the subject

[1] 'The Principles and Methods of Medical Observation and Research.' Edit. 2 (Edin. 1864), p. 338.

[2] 'Lancet,' vol. ii. (1872), p. 115.

of megrim. Discussing the alliances, and the various symptomatic forms, of megrim, he observes that—"There is much in the history of gout—its hereditary character, limitation to particular ages and sexes, periodicity, explosive character, sudden translations and remarkable metamorphic relations with nervous disorders—*which seems to stamp the malady as a pure neurosis;*" and even the fit itself, with its sudden nocturnal invasion, the late Dr. Todd was accustomed to compare to one of epilepsy or of asthma. Moreover, although the presence of uric acid in the blood of gouty subjects is no longer inferential and admits of ready demonstration, the dependence of the remaining phenomena of gout upon this associated condition, is, to say the least, far from proved; and it is further certain that uric acid is also present in excess under other pathological conditions which have no connection whatever with gout. On the whole, there is much to be said in support of the view that *gout in its various forms is the manifestation of a disorder which has its primary seat in the nervous system itself;* and there is no more difficulty in conceiving that inflammation and pain may be an effect of deranged innervation in the case of arthritis, than in the analogous case of herpes zoster; or that an excess of uric acid should be generated or retained in the system under a similar influence, than that sugar should in the parallel case of the diabetes which follows a lesion in the floor of the fourth ventricle."[1]

As bearing on this subject, Sir James Paget[2] remarks that "disturbance of the nervous system in some form and part may be regarded as a factor in every case of gout. There are reasons enough for thinking that changes in the nervous centres determine the locality of each gouty process, while changes in the relations of the blood and tissues determine its method and effects; and that thus we may explain the symmetries of disease in gout, sometimes bilateral, sometimes antero-posterior, and thus its metastases. But these changes are a part of the pathology of gout which is not yet clinical."[3]

[1] 'On Megrim, Sick-Headache, and some Allied Disorders.' By Edward Liveing, M.D. Cantab. (Lond. 1873), p. 404.

[2] 'Clinical Lectures and Essays.' Edit. 2 (1879), p. 382.

[3] 'Studies of Some Irregular Manifestations of Gout' (1879), p. 93.

In a paper published last year in the 15th Volume of St. Bartholomew's Hospital Reports,[1] I alluded to these views of Cullen, Laycock, and Liveing, and quoted the teaching of Laycock on this subject, showing how he regarded the perverted chemistry of gout merely as an epiphenomenon of a more profound and overruling neurosis; and I ventured to predict that at no distant period Cullen's assertions would be more completely verified and vindicated. Cullen's views have been vigorously combated by Dr. Garrod in his classical work on gout, but I venture to think that with additional evidence now forthcoming, and by the light of other modern doctrines established since Cullen's day, the teaching of that illustrious man, with certain modifications, may not unfairly be sustained at the present time.

Gout has certainly held its place for a long time in humoral pathology, and although Cullen's views tended to overthrow the theory of any special morbid state of the fluids, it does not appear that any marked attention was paid to the nervous part of his ætiology. More stress was laid by his followers upon that portion of his theory which embraced the tone or atony of the system. Cullen held that loss of tone occurred in the extremities, and that this atonic state was communicated to the whole system, but especially to the stomach. He believed that nature restored the lost tone by setting up an inflammatory affection in the extremities.

He further developed from this more fanciful theories, to explain different varieties of gout, such as atonic, misplaced, and retrocedent. Such views as these are hardly perused with patience in these days, and the promulgator of them is naturally regarded as an effete authority.[2] But it is possible, I believe, to rescue from oblivion, and to develop more fully, Cullen's theory as to gout being first, "a disease of the whole system, depending upon a certain general conformation and

[1] Mr. Spencer Wells has called attention to the highly developed nervous system of the gouty, and to the influence of irregular exercise of it, in inducing paroxysms.—'Practical Observations on Gout,' etc. (1854), p. 21.

[2] "The view of the solidists, represented by Cullen, who considered gout to be an affection of the nervous system, has never been able to hold its ground against the various humoralistic theories." Senator, 'Ziemssen's Cyclopædia,' Art. Gout, p. 101, Eng. Translation.

state of body;"[1] and secondly, that it "is manifestly an affection of the nervous system."

The researches of Dr. Garrod seemed to place the modern pathology of gout upon a fresh and secure humoral basis. His facts respecting the uric-acid-excreting function of the kidneys, and the relations between this acid and the disease, have not been controverted, and they constitute a solid advance in medical science, which must ever remain most honourably associated with his name. Still, Garrod, with a degree of caution becoming his scientific position, allows that his views are "in themselves insufficient to explain all the phenomena of gout." He has endeavoured to meet every difficulty which opposed itself to his own theory, and, it must be allowed, with great power and ingenuity. But he has nowhere availed himself of any view which would admit the interposition of nervous influence, and he does not attempt to combat Cullen's particular theory of the specific involvement of the nervous centres. Sir Charles Scudamore, who criticised Cullen's views, also took no heed of the alleged implication of the nervous system, and adopted a humoral theory, starting from the stomach.[2] In more recent times too, the late Dr. Murchison gave in his full adherence to a humoral theory of gout, and expressed himself thus: "I hold that what is called a 'gouty diathesis' always indicates, and is the result of hepatic derangement, and that many symptoms commonly referred to gout, would be more correctly ascribed to disorder of the liver. . . . Gout itself is merely one of the results of lithæmia."[3] The idea that gout was in any way a manifestation of disturbed nervous relations, seems never to have been contemplated by that very thoughtful and philosophical physician.

In a suggestive communication by Dr. Ord,[4] on the relations of uric acid to gout, he offers the view that the malady is a

[1] Op. cit., par. DXXX.

[2] 'A Treatise on the Nature and Cure of Gout and Gravel,' edit. 4 (1823), pp. 10 and 147. Cullen's "third observation" was, that the stomach was the internal part most frequently and considerably affected by gout, yet he repudiated any humoral pathology in connection with it.

[3] 'Lectures on Diseases of the Liver.' Edit. 2 (1877), p. 568.

[4] 'Medical Times and Gazette,' vol. i. (1874), p. 233.

special form of degeneration or want of tissue-organisation in remote, and lowly vascular parts. He believes that the uratic deposits result from either general or local disintegration, and are not to be regarded as significant of their elimination from the blood: further, that the local processes are not dependent on these deposits, and that the latter are not the result of the inflammation. He takes cognizance of nervous influence so far as to affirm that "all authors, in one way or another, admit the direct influence of the nervous system" (which statement I can hardly agree with), and he believes that local gouty "degenerations and inflammation tend to infect the rest of the system through the blood, *and to set up similar actions elsewhere through reflex nervous action.*"

Dr. Ord's theory is therefore mainly humoral, and he is chiefly concerned to combat Garrod's views.

To discuss more in detail the question of the humoral element in the pathology of gout, is not within the scope of the present essay.

It is, without doubt, the case that, hitherto, no theory has been set forth which appears to embrace all the multiform phenomena of gouty disease. The greatest advance of modern research has been to establish the certainty of some special relation to it, in the greater number of instances, of uric acid, and, so far, there is clear warrant for retaining a measure of humoral pathology in our conception of the malady. There is still much to be done in elucidating the causes of the renal inadequacy which must be taken into the consideration of a comprehensive pathology of gout.

The best approach to the line of argument I purpose to take up, will manifestly be to review the special characters of neuroses in general, and then to examine coincidently, how far the well-ascertained features of gout conform to such characters.

Before proceeding to this analysis, I would first assert that gout is something beyond the mere resultant effects of aberrant relations of uric acid; that it consists in something more than a perversion of animal chemistry; that it is not, by any means, to be explained as an outcome of gastric or hepatic distemper; and that it is not the appanage only of the middle-

aged or elderly high-liver and intemperate drinker, because, as is well known, it affects also, sometimes in early life, the high-thinker and the laborious bread-winner. Without doubt, while accepting all (and that is much) that it is good for, one is impelled to look beyond what may be termed the chemical pathogeny of gout. The researches into the nature and functions of the nervous system, as carried out during the past quarter of this century, come to our aid at this stage of our inquiry, and, amongst these, we have learned two main and important points respecting the neuroses in general. The first is, that they may be primary or central, and the other is, that they may be secondary or induced. In other words, it may be averred that a neurosis is implanted, or a tendency to it established, and this shall be handed down, hereditarily passed on, and thus, a diathetic tendency be formed; or, owing to some toxæmic condition or blood-degeneration, a secondary or induced neurosis may be established.

I shall endeavour to sustain the neurotic theory of gout upon this basis.

Representing special conditions, or rather special morbid modes of evolution, of nerve-force, neuroses are implanted in the individual as a part of his intimate nature. They belong to the individual, and are characteristic of him in the same degree that are his features and other physiognomical traits. An implanted neurosis is, as it were, the representative of a morbid physiognomy for the cerebro-spinal axis. A neurosis, then, is a peculiar disposition, or tendency, on the part of the nervous system, or some definite tract of it, towards morbid evolution or manifestation of nerve-functions. It does not necessitate the existence of any coarse disease, directly obvious to the eye, but it is a more or less abiding condition, ready to come into action upon suitable provocation.

It is specially characteristic of neuroses that, being thus primarily impressed upon an individual, they tend to be transmitted by heredity. It has been alleged that the female sex is more neurotically disposed than the male; but facts do not support this opinion thus broadly put forth. Certain neuroses appear to prevail with greater frequency in males, and others in females; and not only so, but in the case of those

that are common to both sexes, the manifestation occurs at different epochs of life.

Thus, some outbreaks of neurotic disorders are seen to occur at the several septennial climacteric periods, at the times of dentition, puberty, and often at the grand climacteric. In this manner, an element of distinct periodicity attaches to neuroses in general.

Further, a most marked feature in all neurotic affections is that of paroxysmal tendency. Thus, there is the abiding element, with proclivity to recurring outbreak.

Again, it is certainly known that a law of alternation, or substitution, prevails in neuroses, and thus we meet with certain affections in the parent or ancestors, and with others, equally neurotic, in the collateral relations or descendants. We thus have to deal with distinct types of nervous impression. These abiding conditions are more or less prone to be excited into activity according to various circumstances.

It is not difficult to understand the course pursued by a neurotic taint once laid down or impressed; but it is not so easy to conceive the original implanting of such a morbid functional tendency. The mischief, however, is constantly originating in individuals, and as constantly undergoing further development, modification, or even repression.

Excessive activity of the nervous system, or of any part of it, as Laycock has shown, becomes a highly-disposing cause of the neuroses. Habitual or prolonged excess comes thus to develop hereditary tendency. Thus, *undue mental labour, gluttony, alcoholic intemperance, debauchery, and other indulged evil propensities in the parent, come to be developed into definite neurotic taint and tendency in the offspring.* Particular examples of this are not far to seek, and amongst them comes out the disorder so widely and variously manifested as gout. According to this view, for which I plead, gout appears as a diathetic neurosis, and a link in the long chain of its phenomena, so long missing, is now forthcoming.

I have already stated that there is clear warrant for retaining, as *part of the pathology* of gout, a humoral hypothesis, and it may perhaps be applied and relegated to its proper place, as follows. Granting that gout in any individual is the outcome

of a central neurotic taint, we have the ordinary manifestations of it more or less severe. This we may term primary or central gout. The tendency may be transmitted or modified, or, conceivably, may be allowed to die out.

In another individual, gout may "grow up" where previously there was no neurotic taint or tendency. A patient is commonly said to earn his gout by high-living and over-indulgence of appetites. In this instance a morbid blood-state is induced; excess of uric acid is generated, and hyperinosis supervenes.

But is this all? Is this enough to generate all the phenomena we recognise clinically in gouty disease? I believe not. We are compelled at this point to widen our view, and are driven, perforce, to invoke the operations of the nervous system. Having arrived thus far at nothing beyond a special toxæmia, we must drop humoral pathology, and seek for the effects of the blood-dyscrasia upon the nerve-centres. And we have full warrant for this course in contemplating the analogy of other toxæmic states, together with their effects upon the nervous system. The nutrition of this system is plainly affected by morbid blood-conditions, and, thus, expression is given to such poisoning in the form of convulsion and other nervous symptoms.

I believe, then, it may be conceded that a secondary affection of some nerve-centre occurs as a consequence of the altered blood-state *ab intra*, and that thus the order and particular process of the gouty attack is evolved. This we may term secondary or acquired gout. A diathetic neurosis is thus impressed upon the individual.[1]

It is certainly a matter of much interest to study side by side with gouty processes the several joint-affections, or arthropathies, which have come to be regarded of late as of distinctly spinal or otherwise nervous origin. It seems impossible to separate gouty arthritis from this connection. And if it be conceded that this particular form, which is but one

[1] The frequency and severity of gout in England is explicable on the view of the impressed neurosis. The habits leading to gout,—high-living, intemperance in strong drinks (malt-liquors and wines), along with much mental energy, have certainly prevailed more, and amongst larger classes, in England than in either Scotland or Ireland.

of many others, is truly and directly dependent upon nerve-influence, the greatest part of the difficulty in establishing a neurotic theory of gout is forthwith removed. I suppose no greater obstacle has stood in the way of the acceptance more generally of a nervous theory of this malady, than the impossibility hitherto of connecting arthritic disposition with any form of neurosis. So many of the other, and less obvious, manifestations of gout are plainly dependent on nervous influence, that the whole phenomena now appear to fall more naturally into their places.

It is, however, only right to mention here that thoughtful physicians have long ere now conceived the special action of nerve-influence on joints, and of arthritic affection on nerve-centre.[1] The relation sometimes existing between rheumatic fever and chorea is an example in point, as Dr. Liveing has shown.

Much light has been thrown of late upon spinal arthropathies by the researches of Charcot, Ball, Weir Mitchell, and of Dr. Ord. The latter has recently contended for a more scientific revision of our present views upon the pathology of osteo-arthritis or rheumatic gout.

Without elaborate reasoning, Dr. Noël Gueneau de Mussy, of Paris, expressed, some years ago, his opinion that the latter affection was related to gout, as a sort of cousin, but without in any way holding the view that the disorder was a compound of rheumatism and gout.[2]

Dr. Ord's views are not only eminently ingenious, but they accord remarkably with well-observed clinical facts, not hitherto correlated.

As Sir James Paget has remarked, the changes in the nerve-centres which determine the locality of the gouty process, are a part of the pathology of gout which is not yet clinical. They are, therefore, no more than speculative at present, but we gain much from the prosecution of an inquiry in this direction.

With respect to the particular locality affected in the

[1] Liveing, op. cit. p. 247. Weir Mitchell, Charcot, &c.

[2] Mr. Hutchinson believes in a "basic diathesis," for both gout and osteo-arthritis.

arthropathy of locomotor ataxia there is some discrepancy of opinion. Charcot has declared for implication of the anterior cornua of the spinal chord. Dr. Buzzard, however, has not confirmed this opinion, and, guided by the noteworthy frequent association of gastric crises with these joint-affections in this malady, as previously observed by Dr. Ball,[1] has suggested a sclerosing lesion, involving the roots of the vagus in the medulla oblongata, in close relation to some trophic centre that may be localised there, presiding over the osseous and articular systems. And he further indicates the bond that may thus exist between implication of joints and such metastasis as may occur to the heart in rheumatic fever: also the occurrence of hyperpyrexia, which is sometimes present in such cases. We have yet to seek for this hypothetical nutrient centre for joints, but in the meantime we are fairly warranted in widening our view, and in directing attention to the high significance of predicating such a trophic centre.

"Discovery by true analogies is always progressive, . . . one analogy leads on to another investigation and arrangement of phenomena, and another analogy."[2]

It remains now to be shown, more in detail, how the phenomena of gout conform to the recognised manifestations of the neuroses in general.

It can be shown, I believe, that the plea for the neurotic element in true gout is not difficult to establish.

First, there is to be considered the marked tendency of gout to be hereditarily transmitted. This is notorious. The disorder may pass from either parent, and may be mingled with other taints and tendencies passed on from the progenitors. The outbreak may occur in slight or in graver degrees, and may be deferred, overtly, till even the thirteenth climacteric period. Thus, the first plain attacks of gout may not appear till the patient is sixty or over ninety years of age. In all such cases, however, I am convinced that many minor tokens of the disorder have been overlooked in previous years, all of which are sufficiently obvious to the trained clinical eye.

[1] 'Med. Times and Gazette,' vol. ii. (1868); vol. ii. (1869), p. 498.
[2] Laycock, op. cit. 190.

As a rule, the manifestations are prone to occur at definite ages in each sex, most commonly in the fourth decade in men, and in the fifth in women. My own experience seems to show that gout is increasingly frequent in men early in the third decade.

Certain peculiarities attending gouty transmission are deserving of study. Mr. Hutchinson has called attention to one of these in a suggestive lecture published four years ago.[1] He expresses his belief that what is transmitted is not the active gouty dyscrasia itself, but rather a susceptibility to the influence of certain exciting causes, together with some peculiarly disordered condition of the assimilating and excretory viscera, which renders them unable to deal with particular articles of food. Now, this special susceptibility to definite exciting factors is neither more nor less than a nervous peculiarity, of which the chief character is its liability to break away in certain morbid directions,—its *instability*, in short. This is, I submit, the gouty neurosis. Mr. Hutchinson further believes that gout is wont to show itself with greater frequency and in more marked form in the younger than in the older members of a gouty family, the diathesis strengthening in the parent with advancing years. I think I can confirm this observation.[2] Resemblance to the gouty parent has been specially recognised in those of the offspring most distinctly affected.[3] In other members of the family the tokens of gout may be present, but less marked. These facts are, of course, in accordance with ordinary laws of hereditary transmission. Dr. Wickham Legg has called attention to the fact that gout, like hæmophilia, pseudo-hypertrophic paralysis of Duchenne, and some other affections, is not unfrequently found to be transmitted by the female line, although especially manifested in males, the mothers themselves being unaffected.

A noteworthy feature in gouty ailments is their sudden supervention. As in epilepsy, not uncommonly, the patient often feels remarkably well, and realises his sense of *bien être*, before the outbreak suddenly takes place. This euphoria, or

[1] 'Medical Times and Gazette,' vol. i. p 543 (1876).

[2] Cases illustrating this are given by Mr. Spencer Wells, op. cit. p. 18.

[3] 'Medical Notes and Reflections on Hereditary Disease.' Sir Henry Holland, Bt., M.D., F.R.S. Edit. 3 (1855), p. 29.

delusive corporeal satisfaction, is itself a nervous derangement. Explosiveness is a distinct feature in several of the neuroses, and attaches to such ailments as angina pectoris, asthma, epilepsy, and various neuralgiæ.

The time of the occurrence of the attack is also strongly marked. The majority of the outbreaks take place in the early morning. This is true both of grave and classical cases, and also of many of the minor forms of gouty trouble. The same thing is met with in asthma, neuralgia, and in epilepsy. The pyrexia proper to acute gout is paroxysmal, with remissions, and the pain of gout is likewise paroxysmal. One is here reminded of the influence of marsh-poison upon the nervous centres.

This paroxysmal, no less than the periodic, element in gout, stamps a nervous character upon the malady, and binds it in alliance with other neuroses.[1]

An important connection of the same kind is seen in the unquestionable commingling of gout with other well-recognised neuroses. Thus, hemicrania is sometimes distinctly a manifestation of gout in both sexes, and may be the form of neurosis impressed upon an individual whose parent was gouty, or may itself alternate with gouty arthritic attacks in the same person.[2]

It is not far to seek for an allied condition of trophical lesion in herpes zoster, itself the outcome of disordered innervation.

The doctrine of metastasis must next be considered in relation to gout. The humoralist seeks to explain this clinical fact upon his theory, but such is manifestly insufficient to account for the phenomena. It must be conceded that some nervous law regulates the occurrence of shifting inflammation. It has been supposed to be due to reflex influence. Some distinct predisposition to take on the morbid action exists in the part selected apparently by caprice. The same class of tissue is apt to suffer. Thus, the gouty or rheumatic process flies from joint to joint, or, as in gouty phlebitis, from vein to

[1] Vide Scudamore, op. cit. p. 152.

[2] Stahl, op. cit. § xxxvi. Trousseau, 'Clin. Méd.' Liveing, op. cit., &c. Sir H. Holland, op. cit., 'Relation of Asthma to Gout,' p. 36.

vein, sometimes symmetrically, but not always. The serous and fibro-serous structures suffer especially, but also mucous surfaces. Laycock has shown how these several tissues are related embryologically, and are thus prone to suffer in common when diathetically impressed.[1]

Localised trophical changes follow locally acting causes of depressed nervous power. Thus, impairment of certain centres may lead to the specific nutritional changes witnessed in metastases, and thus the apparent capriciousness is explained in this process.

Amongst the nervous symptoms of gout must next be considered the occurrence of certain sensory perversions, such as tingling and numbness of the fingers and toes, sensations of heat in the palms, thighs, and soles (paræsthesia), and tickling in the throat. As pointed out by Sir James Paget, "gout affects the sensory much more than the motor elements of the nervous system," and he remarks, too, that the pain of acute gout is seemingly out of all proportion to the amount of inflammatory process in the affected part. So, too, all other disorders, modified by gout, seem to be especially painful, for example cancer, as pointed out also by Paget.

Grinding of the teeth is met with in the gouty. Graves first observed this.[2] Garrod has not met with it. Dr. Donkin has lately recorded cases associated with somnambulism,[3] and I have intimate knowledge of two others in which the same phenomena are manifested—the grinding of teeth and somnambulism in a sister, and the talking in sleep in the brother. The maternal grandfather and the mother are distinctly gouty. Cramps in the muscles of the legs, and priapism, are amongst nocturnal manifestations in the gouty. Of insomnia, due to gout, there is much to say. It was originally noted by Cullen, and it conforms remarkably with other periodic neurosal phenomena.[4]

Gouty neuralgia is largely recognised, and is known to be both severe and prone to recur. It is frequently occipital,

[1] Op. cit., p. 196.
[2] 'Clin. Med.,' p. 351 (1864 edit.).
[3] 'Brit. Med. Journal,' Feb. 21, 1880, p. 279.
[4] Vide Author's paper, 'St. Barth. Hosp. Reports,' jam cit., p. 105.

and is met with in the heel, tongue, breast, and more often in the great sciatic nerve. One proof, amongst others, of the truly gouty nature of these is gained from the fact that they yield most readily to anti-gouty medication, and another lies in the frequency with which they are provoked by conditions which elicit other gouty processes.

Amongst the strongest evidences of gout depending upon nervous influences, are the unquestionable facts bearing upon the induction of its attacks.

The influence of many of the existing causes of gouty paroxysms illustrates well the explosive character of the malady. As Sydenham expresses it, before the onset of an attack, "totum corpus est podagra." The precipitation of the seizure sometimes ensues almost immediately upon the provoking cause. In a large number of instances, the latter is of a nature *to depress nervous power*. Thus, unwonted muscular energy, prolonged exercise, stirring emotions, fright, undue excitement, venereal excess, rage, worry, and vexation, are all excitors of gouty paroxysm. So, too, sudden shock to the body, as from injuries and surgical operations, will evoke gout. Dietetic errors are well recognised as factors; thus, a full meal, and excess, or mixing, of strong liquors, will act in upsetting the equilibrium of a quiescent gouty habit. It will be conceded that many of the causes just enumerated are equally potent to elicit manifestations of other neuroses, such as epilepsy, asthma, hemicrania, and angina pectoris. The provoking agency, however, need not always be primarily exhausting. In proof of this, the outbreaks of gout following hydropathic treatment, internally or externally, may be instanced, and, indeed, the causal element need only be such as shall induce some change in the acquired vital habits.

Thus it is, that the subjects of the neuroses hold much of their comfort in life by following a very equable routine. They are prone to give way under any extraordinary pressure.

These considerations explain, in part, why men are more liable to gout than women. They carry on the world's rough work; are engaged in more exciting occupations, and have commonly the greater burden of anxiety to bear.

The more sedentary the occupation, the more profound

the mind-working, and the more intense the strain of life, the greater the tendency to nervous depression, and to the peculiar form of its expression in gout. If to such habits be added high-living, as often occurs in the cases of eminent statesmen, lawyers, and speculators, no link is wanting in the chain of causation, and all the elements for gout are present.

Climatic influence is important amongst these agencies. The dull and "shifty" weather, and the cold east winds of northern latitudes are certainly bad for gout. The nervous depression ensuing upon months of sunless skies—negation of light powerfully lowering nervous tone—is too little regarded as an element of devitalisation in England. The cutaneous eliminant power is checked, and so, aberrant chemical relations are apt to be determined in any parts specially prone to gouty invasion.

The same mal-determination ensues upon the suppression of various discharges, whether from the uterus, from hæmorrhoids, or other sources.

The mental condition of the goutily-disposed is a subject worthy of attention in relation to the pathology of the ailment.

Hypochondriasis has long been associated with gouty taint. It commonly precedes an outbreak, and disappears subsequently. A tendency to sighing has also been observed, and is a plain indication of nervous exhaustion. Hysteria has also been observed to precede gouty attacks in women, and to disappear with the onset of articular symptoms.[1]

Irritability of temper is a proverbial condition in the gouty, and furious outbursts of this kind appear to be, at times, a metamorphic substitution for a more overt and regular attack. It is important to know that many of the minor, but none the less well-marked, phases of gouty paroxysm are in no degree arthropathic. Much error in diagnosis has arisen from taking no heed of any but articular symptoms when searching for gouty tokens in a given case. These less classical attacks very commonly precede the onset of typical ones at a later period in life.

The necessity for prompt recognition of these less well-

[1] 'On the relations between Gout and Hysteria,' *vide* 'Treatise on the Nervous Diseases of Women' (p. 163). By T. Laycock. 1840.

expressed symptoms is obvious, if good treatment is to be applied.

Epilepsy has been known to disappear on the supervention of gout.

Sensations of giddiness and dimness of vision, not uncommon in the gouty, are noteworthy in relation to nervous symptomatology.[1] So, too, the disturbances of the cardiac rhythm, and the co-existent (neurotic) vascular throbbings which are sometimes met with. The cardiac irregularity has been noted to cease with the induction of a regular attack. Dysphagia was noted in connection with gout by no less careful an observer than the late Dr. Brinton.

A consideration of the effects of lead-impregnation in relation to gout, and of the certain liability of the gouty to be more readily than others influenced by lead, leads to the belief that the nervous system is specially implicated in these relations. The fact is, that the blood is imperfectly freed from uric acid in cases of lead-poisoning, and that gout is thus quickly evoked. Dr. Garrod has fully established these facts, and all physicians now recognise them. The lead-influence, clearly through nervous agency, induces the measure of renal inadequacy which is, probably with correctness, acknowledged as a factor in gout. And the knowledge that this metal is capable of setting up special paralysis, epilepsy, coma, and other cerebral phenomena, is of the largest interest in relation to this subject.

I now approach a point in connection with the whole pathology of gout which merits much consideration. The connection of diabetes with gout has been recognised for some years.

I object to the term diabetes as applied to the special form of glycosuria associated with gout. The disorder is met with in certain members of gouty families, some having regular gout, and others manifesting less regular gout, or this alternating with glycosuria. I believe that many cases of temporary glycosuria are due to gouty conditions. The fleeting presence of glucose in the urine of many elderly persons may be thus explained. It has long been recognised that such an affection, which, in many instances, is undeserving of the name

[1] Trousseau, Murchison, Paget, op. cit., H. Mayo, F.R.S., 'Philosophy of Living,' p. 24 (1837).

diabetes mellitus, for the simple reason that there is no *diabetes* in the strict sense of the term, is not really a grave one. *The presence of glucose is found to alternate with that of uric acid.* In the aged but little importance should be attached to the symptom. Charcot testified to this, some years ago, in his excellent lectures on the maladies of old people, his experience being gathered at that fertile school of study—the Salpétrière. In persons under forty years of age, however, glycosuria, even of gouty origin, is a most grave matter, and merits the closest attention, since it may eventuate in confirmed diabetes. It is the rule to find that the quantity of urine passed is not much, if at all, above the normal, but the specific gravity may range from 1·035 to 1·050. An anti-gouty treatment is called for, for the glucose may otherwise only give place to uric acid or increased azoturia, and the gouty habit has rather to be attacked than the glycosuria.

Dr. Lauder Brunton has called attention to this class of cases.[1]

The alliances of gout and diabetes are sufficiently intimate. In both the doctrine of heredity applies, and the nervous system is involved. The same habits lead to each, the same classes of person are affected, and the same exciting causes are potent to evoke both. A consideration of these facts naturally leads to the belief that the portions of the nervous system involved in each cannot be far apart from one another. The medulla oblongata, the sympathetic and splanchnic nerves have been found chiefly affected, and the spinal chord likewise in some instances. The point for the diabetic puncture in the medulla is believed by physiologists to correspond to the vaso-motor centre in the same structure.

Guided by these facts, and by the knowledge that the glycogenic function of the liver is under nervous influence, by the advancing theories which refer special arthropathies likewise to the same influence, and bearing in mind Dr. Buzzard's views, previously stated, in connection with the gastric crises so commonly associated with the arthritis of loco-motor ataxia, I come to the conclusion that the portion of the nervous system which is specially predisposed to the irregular mode of action known as gout, has its seat or centre in the medulla oblongata.

[1] Art. "Diabetes Mellitus," Reynolds' 'Syst. of Med.,' vol. v. p. 381 (1879).

A point of difference between the arthritic affections which are now referred to nervous influence, and those manifested in gout, is found in the fact, that the latter appears to have an elective affinity, often unilateral at first, for the smaller joints, especially that of the big toe, while most of the others influence the larger ones. Herein, perhaps, lies part of the specificity of gout.

The trophical results of the latter are seen impressed upon the physiognomy, and upon certain tissues, in a manner extremely definite and characteristic.

Thus, are found the large head, the thick hair, with tendency to early greyness, the large, full veins, the long uvula, the soft, smooth skin, the thickened extremity of the nose, and the lineated, brittle nails.

Lastly, I may add an argument in favour of the theory, adduced in this essay, from the therapeutical side.

The universally acknowledged specific action of colchicum in gout is known, owing to Garrod's researches, to be due to no power which it possesses of causing elimination of uric acid. Gouty inflammation is therefore influenced by it without reference to the secondary aberrant relations of uric acid. The active principle, or alkaloid, of the drug colchicia, is a member of a nitrogenised group of bodies to which veratria, strychnia, quinia, and morphia have close chemical alliance.[1] They all powerfully affect *the nervous system*. Colchicum acts very promptly, and affords often decided relief to the intolerable pain of the gouty process. When taken in health, in small doses, Dr. Meldon and others have found that it induces a general glow at the surface of the body, diaphoresis, throbbing of the blood-vessels and palpitation. Subsequently, there is reduction in the force and frequency of the pulse. Dr. Meldon observed in his own case an invigoration of his mental energies. In larger doses, the effects are most marked along the whole tract supplied by the vagus, and thus, cardio-vascular, gastric and enteric symptoms ensue.

The peculiar benefit derived from this drug is not secured in any other form of inflammation, and thus it is plainly

[1] Vide 'Lectures on Pathology and Therapeutics.' (London, 1867; p. 137.) H. Bence Jones, M.D., F.R.S.

specific. Its cherished action is doubtless exerted upon the vaso-motor nerves.

The manifestly good influence of all agencies which cheerfully inspire the mental condition in the goutily-disposed, must not be omitted from consideration amongst the *juvantia* both of prevention and cure.

The points in this thesis which I have endeavoured to sustain, I now place categorically under the following heads.

First.—I contend that the diseased conditions which are recognised as of unequivocal gouty nature, are primarily dependent upon a functional disorder of a definite tract of the nervous system, and that, thus, gout is a primary neurosis.

Secondly.—That there is much in the nature of the malady itself, and much evidence forthcoming by way of analogy, to warrant the conjecture that the portion of the nervous system specially involved is situate in some part of the medulla oblongata, where, possibly, may be placed a trophic centre for the joints.

Thirdly.—That the gouty neurosis may, like others, be acquired, intensified, and transmitted, also, that it may be modified variously, and commingled with other neuroses; that it may suffer metamorphic transformations, or be altogether repressed.

Fourthly.—That this diathetic neurosis imposes its type upon the affected individual in definite nutritional modes, affecting the assimilating and excreting powers, exhibiting marked peculiarities in nervous impressibility, and determining, in more or less degree, a physiognomy of the gouty.

Fifthly.—That a large part of the phenomena known as gouty, are due to perverted relations of uric acid and sodium salts in the economy, resulting from the morbid peculiarities mentioned under the last head. Thus, there is excess of urate of soda in the blood before, and during, gouty explosive manifestation, and there is determination (by nervous influence, in all probability) either of this salt to the affected part (Garrod),[1] or there is a too free formation of it at these inflammatory

[1] Op. cit.

points, whence it deposited locally, and also set free into the circulation (Ord).

The renal excretory power for uric acid appears to be temporarily inhibited as part of the process of gouty paroxysm. This measure of renal inadequacy would appear to prevail in varying degree as a part of the specific neurosal disorder. In chronic gout, when structural disease has occurred, either tubal, with deposition of urate of soda, or interstitial, with shrinking of the organs, the renal inadequacy may admit of more mechanical explanation.

Sixthly.—That in Primary, or inherited, Gout, the toxæmia is dependent on the gouty neurosis; is the outcome, in whatever degree, of it, and is therefore a secondary manifestation.

Seventhly.—That, in what I term Secondary, or acquired, Gout, the toxæmia is directly induced by such habits as overload the digestive and excretory organs, and constantly prevent complete secondary disposal of nutritional elements of food; that if, together with such toxæmia, distinctly depressing and exhausting agencies, affecting the nervous system, come into operation, the special neurotic manifestations of the gouty diathesis will occur, and be impressed more or less deeply upon the individual and his offspring.

Eighthly.—That this theory of gout, better than any other, correlates all the known factors concerned in the production of the varied symptoms of the malady; and while it displaces its humoral pathology from the preeminence it has so long occupied, it takes full cognizance of it, and seeks to place it in a clearer relation to the phenomena of the disease.

Ninthly.—That if it be desirable to refer various maladies to their distinct place in pathology, without reference merely to their chemistry, histology, or neurology, the affection known as gout may perhaps most correctly be relegated, along with some others, to a class of diseases which may be termed neuro-humoral.

Tenthly.—An argument is adduced from the *juvantia* afforded by colchicum, in favour of the theory which has been set forth.

THE CONDITIONS OF THE UNIPOLAR STIMULATION IN PHYSIOLOGY AND THERAPEUTICS.

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By electrotonus is meant that condition of altered irritability of a nerve which is produced by the flow through it of a current of electricity; and this altered irritability manifests itself by increased or diminished reaction of the nerve to the various stimuli, whether mechanical, physical, or chemical, which may be applied to it.

Du Bois Reymond, who introduced this word in physiology,[1] at first implied by it merely the changes in the electromotive manifestations of the nerve which accompany the changes in its irritability. A clear recognition of the latter, says Hermann,[2] does not occur in any of the older physiologists, nor in Du Bois Reymond's great work. Valentin[3] was the first to show that the polarised portion of the nerve does not readily transmit impulses from above; and that the irritability of the part below it is diminished when the polarising current is centripetal, or ascending. Eckhard[4] observed that when the current is descending, an opposite condition of augmented irritability below the polarised portion existed. Hence he formulated as a general law that irritability is increased beyond the kathode, or negative pole; diminished beyond the anode, or positive pole.

Pflüger, by a series of admirable experiments, eliminated all the sources of error which beset this difficult field of inquiry, and resolved all the phenomena into his dictum:—The irri-

[1] 'Untersuchungen über thierische Elektricität,' 1848, 1849.
[2] 'Handbuch der Physiologie,' vol. ii. part i. p. 41, 1879.
[3] 'Lehrbuch der Physiologie,' 2nd Ed., 1848.
[4] 'Beiträge zur Anatomie und Physiologie,' 1855.

tability is increased on either side of the negative electrode, diminished on either side of the positive. Or, in other words, the region of the kathode is thrown into a state of katelectrotonus, that of the anode of anelectrotonus. In the intra-polar region the two zones meet at a point of indifference, where irritability remains normal. This point is the nearer to the kathode the stronger the current, and *vice versâ*.

Whenever a galvanic current of sufficient strength is made or broken through a portion of a motor-nerve, a contraction in the muscle occurs. It was one of the earliest observations made that a contraction appears earlier (i. e. with a weaker current) when the current is descending (i. e. with the kathode peripherally and the anode centrally placed) than under the opposite conditions (Pfaff, in 1795). Ritter (1798), and Nobili (1829), with a host of other observers, confirmed this fact, and elaborated what are known as "laws of contractions;" that is, tables of the reactions which the exsected nerve yields to make and break off the ascending and descending currents, at different stages of its diminishing vitality. That such researches must have been limited is made at once evident by the consideration that the first constant galvanic element was invented in 1836 by Daniell. Yet on the whole it is possible to reconcile the main results obtained up to the time of Du Bois Reymond,[1] with those of Heidenhain,[2] who substituted "stages of current strength" for the "stages of irritability" of his predecessors. But it would be transgressing our present limits to delay on these topics, and we at once reach Pflüger, to whom we owe also the expression of the phenomena of nerve-reactions to different current-strengths in their simplest formula :—

Current.	Ascending Current.		Descending Current.	
	Make.	Break.	Make.	Break.
Weak	Contraction	Rest	Contraction	Rest
Medium	Contraction	Contraction	Contraction	Contraction
Strong	Rest	Contraction	Contraction	Rest (or weak C.)

[1] Loc. cit. [2] 'Archiv für physiologische Heilkunde,' 1857.

Pflüger did not remain content with leaving his law of contraction and his law of electrotonus side by side; but by a further induction reduced the one to the other, and showed in the happiest manner that a nerve is stimulated by the apparition of katelectrotonus, and the disappearance of anelectrotonus; that is, by the passage of a lesser to a greater degree of irritability. In other words, that the stimulation occurs at one electrode only: at the kathode on closing, at the anode on opening the current. He completely exposed the fallacy of the views of the previous writers, who had all explained by the differences in the direction of the current itself the different results they obtained by the making and breaking of ascending and descending currents; and showed that it was the relative position of the two poles *to one another*, and not to the direction of the nerve-impulse, that was the condition upon which depended the variations in the results obtained.

His argument was based on the facts that the closure of the current is a more powerful stimulant than the opening; that anelectrotonus takes more time and stronger currents to develope than katelectrotonus; and that immediately after the opening of the polarising current the katelectrotonic zone passes into a condition of transitory diminution of irritability, or "negative modification," whilst the anelectrotonic zone passes immediately into one of increased irritability, or "positive modification." Thus it becomes clear why weak currents excite the nerve at the closure only: anelectrotonus is not sufficiently developed by them to cause a contraction on its disappearing, nor to prevent the transmission of the katelectrotonic stimulus from above. Strong currents, on the other hand, develop a powerful anelectrotonus, and negative variation, which arrest the make, and break impulse from above respectively. Medium currents allow a sufficient anelectrotonus to set in which will stimulate on disappearing; but not to arrest the central katelectronic impulse, whilst the negative modification of the katelectrotonic region also is too weak to arrest the stimulation produced by the disappearance of anelectrotonus on the breaking of the descending current.

In presence of the clear fact that, according to Pflüger, stimulation is produced at the kathode on closing, at the anode on

opening of the current, and at the former more readily than at the latter, it is difficult to see how many electrotherapeutical writers have been able to continue speaking of the results obtained by physiologists as dependent on the direction of the current with reference to the nerve; and to place their so-called "polar method" in a position of antagonism to the "physiological method."[1] In the latter, it is true, in order to eliminate many disturbing elements, such as the influence of the central organs, the action of derived currents, &c., the nerve is usually exsected, and both poles applied to it directly; but apart from these differences of manipulation, there is no such fundamental difference between the two methods as has been claimed. Let us first throw a retrospective glance into the origins of that polar method, the subject of so many misunderstandings and passionate recriminations.

Among the earlier observers we come across but few and vague data concerning the effects of the galvanic current on the living body. Ritter[2] noticed that a strong current flowing from hand to hand produced in the arm up which it flows a sensation of increased mobility, and *vice versâ*. Matteucci[3] repeated the observation of Nobili, that tetanised frogs' legs were quieted by an ascending current, and even proposed therapeutical applications of this fact.[4] Valentin[5] is the first to make an explicit statement on the subject. He finds that in the living animal contraction occurs most readily on closing the current, whatever be its direction. Fick and Orelli[6] found the same happen in their experiments on the ulnar nerve in man. Schiff[7] puts down as a law that "contraction occurs in the living subject on making the current in

[1] Hermann says (loc. cit. vol. ii. p 63): "Pflüger the first showed that stimulation of the nerve occurs at one electrode only, on making the current at the kathode, on breaking it at the anode"; and recognises Chauveau's independent discovery of the same fact. Biedermann, by the way ("Uber die polaren Wirkungen des elektrischen Stromes im entnervten Muskel," 'Sitzungsberichte der K. K. Akad. d. Wissenschaften,' vol. lxxix. p. 289), has successfully demonstrated the polar effects on curarised muscle.

[2] 'Beiträge zur nähere Kenntniss des Galvanismus,' Jena, 1802.

[3] 'Essai sur les Phénomènes électriques des Animaux,' 1840.

[4] Cf. 'Traité des Phénomènes electro-physiologiques,' chap. ix., "Usage thérapeutique du Courant continu." 1844.

[5] Loc. cit.

[6] 'Wiener Medicinische Wochenschrift,' 1856.

[7] 'Lehrbuch der Nervenphysiologie,' p. 80, 1856.

either direction, but no contraction on breaking it," adding, it is true that in his experiments he never closed the current for more than five seconds. Claude Bernard, whose classical lectures appeared at the same time,[1] says that "a nerve placed in normal organic conditions, fit to transmit voluntary impulses, gives only one contraction, which occurs on closing the current, whatever be its direction," adding that deviations from this law depended on the mutilation of the nerve.[2] This point has been lately taken up by Rumpf,[3] chiefly from a clinical point of view. He shows that the apparition of the A.O.C. is delayed by the influence of the central organs and hastened by the removal of this influence. There is thus a qualitative disturbance of the normal galvano-nervous formula, which has escaped notice hitherto, owing, probably, to the shortness of the period at which it is recognisable—a few days only, immediately after the injury. The subject deserves further investigation, but much care is required to avoid fallacies. It is probably owing to the influence of Remak[4] that electro-therapeutists have had so much difficulty in realising the full bearings of Pflüger's conclusions. Remak's researches on the effects of the galvanic current on the nerves in the living subject were mainly directed to the phenomenon of "galvanotonus," that is, of contraction during the continuous flow of the current. This he found to be more marked when the current was descending.

Grapengiesser, as early as 1801,[5] had expressed himself clearly to the effect that the negative pole was the more effectual of the two, both at the closing and during the passage of the current. Remak takes extraordinary pains to show that this superiority of the kathode, due to its chemical properties, exerts no influence on the results of the transverse stimulation of nerves. If, for instance, the two poles are

[1] 'Leçons sur la Physiologie du Système nerveux,' vol. i., 1858.

[2] Similar observations were made by Pflüger, Bezold, and Rosenthal. See Meissner's 'Bericht' for 1858. Romanes (Proc. Roy. Soc., 1876 and 1877) has measured the changes in the excitability of the frog nerve produced by injury. Thus, before section A.C. = 90; K.C. = 100; A.O. = 14; K.O. = 6; whilst after section A.C. = 140; K.C. = 300; A.O. = 195; K.O. = 14.

[3] 'Archiv f. Psychiatrie,' vol. viii. p. 567.

[4] 'Galvanothérapie,' 1859.

[5] Quoted by Remak, loc. cit., p. 102.

placed on the temples, it is noticed that contractions are more readily obtained on the side of the kathode; but this is probably due to the fact that the current there is "ascending," for the current avoids the resisting bones, and passes round about the forehead. Even were this not the case the phenomenon could be readily explained by the assumption that a nerve is more readily stimulated by a current flowing transversally through it from its inner to its outer aspect than in the opposite direction![1]

The method usually spoken of in electrotherapeutics as "Brenner's polar method" was, so far as its physiological basis is concerned, fully described by Baierlacher in 1859.[2] Before entering upon this subject, I may be allowed to offer a few remarks on the impropriety of the word "polar." By it is implied that the method rests upon a recognition of polar, instead of directional, influences, and is meant to distinguish it from the so-called "physiological" method. But the truth is, that, as we have already stated, in physiology, since Pflüger, it is to the specific action, chemical or otherwise, of each pole that the effects of the current are attributed. The physiological is, therefore, a polar just as much as the therapeutical method; the first is *bi*polar, the other *uni*polar. By these terms we simply mean that either both poles or only one pole is applied to the nerve. I need hardly point out that the term unipolar, taken in this sense, has nothing to do with the unipolar effects of induction discovered by Du Bois Reymond in 1845,[3] and since then studied by a large number of observers.[4]

In order to let the reader judge for himself how far Baierlacher had anticipated Brenner, I quote some abstracts:—

"The unipolar method of nerve-stimulation adopted by me gives us the opportunity of observing the separate action of each pole on the nerve, whereby we attain the peculiar conclusion that the same phenomena are produced which we are accustomed to consider as depending upon the direction

[1] Sounder views, however, prevail in Remak's later writings, e.g., in his 'Leçons sur l'Application du Courant continu.' Paris, 1865.

[2] 'Zeitschrift für rationelle Medicin,' series III., vol. v., p. 233. Compare Meissner's abstract in 'Bericht,' ibid., vol. vi. p. 442.

[3] 'Untersuchungen,' vol. i. p. 423.

[4] See Hermann, loc. cit., p. 86.

of the current. The positive pole on the nerve gives us the results of the 'ascending,' the negative of the 'descending,' current."[1]

The nerve chosen by Baierlacher for his first experiments was the peroneal, near the head of the fibula; and the result of numerous experiments was that,[2] "with the negative pole on the nerve, the closure contraction was very strong, the opening contraction absent, or exceedingly feeble. With the positive pole to the nerve, the opposite took place, the closure contraction being absent or very weak, the opening contraction strong." Thus the order was K.C.C., A.O.C., A.C.C., K.O.C.: a more accurate statement than that of Brenner, who places A.C.C. before A.O.C., owing probably, as we shall see, to a want of care in applying his electrode.[3]

The author further states[4] that "there can be no question about a difference in the direction of the currents" in his experiments, as the contractions appeared in the same order whether the indifferent electrode was placed above or below the other. He displays considerable acumen in the remark that, even with these changes in the position of the indifferent electrode, the upward or downward direction of the current can be but of secondary influence, compared with that exerted by the very different densities of the current at its points of entrance into, and exit from, the nerve respectively.

Baierlacher also repeated the experiment of Fick with both poles on the ulnar nerve, and came to the conclusion that the order of contractions was—

Current.	Descending.	Ascending.
Closure.	Very Strong.	Strong
Opening	Absent or Weak	Moderate

A result which coincides with the view that the anodal reactions of the unipolar method correspond with the "ascending," the kathodal reactions with the "descending," reac-

[1] Page 253. [2] Page 249.
[3] Or, at least, to his using a too large 'different' electrode. [4] Page 251.

tions of the bipolar method. This point is recognised by Meissner.[1]

Chauveau, at the same time, following an entirely different train of thought, came to similar conclusions: a result all the more meritorious that he worked in complete ignorance of all that was being done in Germany, and laboured under serious misconceptions, such as an imaginary influence of electrical "tension" and of "extra-currents." His experiments[2] were made with Leyden jars, induction coils, and galvanic batteries. The two former bring about contractions more readily at the negative than at the positive pole. On making a weak galvanic current, stimulation occurs at the negative pole only; the direction of the current has no influence whatever. For instance, when the poles are on the sciatic plexus and the lower part of the sciatic nerve, contractions occur in the thigh and leg when the cathode is on the plexus; in the leg only, when it is on the nerve.

The opening contraction occurs, under similar conditions, at the positive pole. In every case the indifferent pole may be applied to any part of the body without changing the results. Space does not allow me to indicate more than the general results of Chauveau's elaborate papers.[3] A good abstract of his researches has been given by Meissner, who adds[4] that these results coincide with those of Baierlacher,

[1] Loc. cit.

[2] "Théorie des effets physiologiques produits par l'électricité transmise dans l'organisme animal à l'état de courant instantané ou de courant continu," 'Journal de la Physiologie,' 1859, pp. 490, 553, 1860, pp. 52, 274, 458, 534, ff. See also Meissner's 'Bericht,' in 'Zeitschrift für rationelle Medicin,' 1860, p. 554.

[3] I cannot here pass under silence Chauveau's latest contributions to the subject of unipolar stimulation. ('Comptes Rendus de l'Académie des Sciences,' 1875, 1876, vols. lxxxi. pp. 779, 824, 1038, 1193, and lxxxii. p. 83). In a series of notes to the Academy he has formulated a number of propositions which are mostly in direct contradiction to all the received views, and his own former results. His recent experiments have been made both on frogs' and mammalians' nerves. To discuss them here is impossible, as they are but briefly described; but they will have to be carefully controlled. He reaches the astonishing conclusion, that for every motor-nerve there exists a current-strength (usually very weak) which gives to both poles the same degree of activity. Below it is the negative pole, above the positive, which preponderates. For sensory nerves it is the opposite that holds.—What precedes applies to the *closure* of the current. On opening also, Chauveau finds the positive pole the more active. His results are embodied in a number of tracings and tables of curves.

[4] Loc. cit., p. 455.

and with Pflüger's theory that stimulation is produced at the appearance of katelectrotonus (= closure contraction when the kathode is on the nerve), and at the disappearance of analectrotonus (= opening contraction when the anode is on the nerve).[1]

The untiring zeal and brilliant success of Brenner in applying the unipolar method to the practice of electro-diagnosis, his success in upsetting the obsolete views of Remak and his followers,[2] are quite sufficient to secure him a prominent place in the history of the question; though Pflüger's law, rightly understood, on one side, and the results of Baierlacher and Chauveau on the other, deprive him of the priority which seems generally to be conceded to him. It was only in 1862[3] that he first published his "discovery" that the closure contraction depended upon the negative pole, the opening contraction upon the positive. In his great work[4] he has collected a vast array of experiments and illustrations in support of his views. The first volume is devoted to the application of the unipolar method to the acoustic nerve, which gives a very pure series of reactions in the normal condition, responding to the cathodal closure (and duration) and anodal opening only. He endeavours to base a rational system of electro-otiatrics on this result. In the second volume he describes the apparatus and manipulations neces-

[1] I think I may fairly claim as supporting the main argument of this paper the results obtained by Morat and Toussaint ('Comptes Rendus de l'Académie des Sciences,' 1877, vol. lxxiv., p. 503), with reference to the electrotonic state of nerves excited on the unipolar method. These observers found that the modifications were of the same order on both sides of the point of application of the electrode. When the kathode was applied, the modification was positive at both ends; when the anode, the modification was negative. Now, in the usual bipolar application, the modification at each end is of the same name as the neighbouring pole. The authors explain the results of the unipolar excitation by saying that the current follows the nerve in two directions; opposed to the natural nerve current in one case, concordant with it in the other; hence the negative and positive modifications. But is it not simpler to assume that it is the virtual anodes or kathodes, on either side of the actual electrode, which produce the phenomenon? When the positive pole is on the nerve, the two virtual kathodes will naturally call forth the negative modification at each end; and the opposite will happen when it is the negative pole which is in contact with the nerve.

[2] See, for instance, Benedikt, 'Elektrotherapie,' 1st Edition, 1866, so unsparingly criticised by Brenner in his 'Untersuchungen,' vol. ii. p. 208, ff.

[3] 'St. Petersburger Medicinische Zeitschrift,' vol. iii.

[4] 'Untersuchungen und Beobachtungen auf dem Gebiete der Elektrotherapie,' Leipzig, 1868–69.

sary to operate successfully, discusses the relations of the "polar" to the "physiological" methods, and makes a substantial contribution to the art of electro-diagnosis.

The effect of Brenner's work was to call forth a controversy as violent as it was useless, but it stimulated also the zeal of a large number of workers in the field. Though the din and dust of the battle caused the loss of much time and temper, there gradually arose clearer views, and valuable results were obtained. The question of the possibility of demonstrating the phenomena of electrotonus in the living human nerve occupied the attention of several observers. First, Fick,[1] whose results were negative, then Eulenburg[2] and Erb[3] appear in the field. Eulenburg, applying the galvanic current to an accessible nerve, such as the peronæus, ulnar, &c., tested its irritability by means of an induced current below the point of application. His results were in accordance, apparently, with those obtained on the exsected frog's nerve; and he found increased or diminished irritability when he produced descending extrapolar katelectrotonus or anelectrotonus. Erb was not so successful in his first experiments, and found a complete inversion of the phenomena, though he followed a method similar to Eulenburg's. Helmholtz[4] thereupon made an important suggestion, which seems to me to contain, as in a germ, the solution of the main difficulties encountered in the experimentation on the living body, and which will be developed further on. He said that Erb's paradoxical results might be due to the fact of the electrical diffusion, which in the immediate neighbourhood of the electrode must be sufficiently considerable to justify the assumption that it there acts as an opposite pole. Acting upon this supposition, Erb tested the condition of the nerve immediately under the electrode, and found it coincide with that obtaining in physiological experiments. The results of Samt[5] are interesting

[1] 'Medicinische Physik,' 1866.

[2] "Ueber elektronisirende Wirkung bei percutoner Anwendung des constanten Stromes auf Nerven und Muskel," 'Deutsches Archiv für klinische Medicin,' vol. iii. p. 117 (1867).

[3] 'Ueber elektrotonische Erscheinungen am lebenden Menschen,' ibid. p. 513; and 'Galvanotherapeutische Mittheilungen,' ibid., pp. 238 and 333.

[4] In an oral communication to the Naturhistorisch-Medicinisch Verein meeting at Heidelberg, 1867.

[5] 'Der Elektrotonus am Menschen,' Berlin, 1868.

chiefly on account of his conclusion that many of the inconsistencies among the results obtained on the human subject are due to morbid conditions of the nerves: a view supported by the fact that the gradual exhaustion of a frog's nerve is a frequent cause of fallacy in experiments, and that the ventral organs exert a marked influence in the time of apparition of the several reactions.

Brückner's results[1] do not throw much further light upon the subject; but the method he used suggested to Runge[2] a series of experiments which are described in a very able paper. This method consisted in intercalating in the circuit of the polarising current the secondary coil of the testing instrument. Brückner found that when the two currents moved in opposite directions the effect was diminished at the negative pole of the induced current especially. Runge performed a series of ingenious experiments, the result of which goes far to prove that in all anterior electrotonic experiments the summation of effects of the two currents must be the real cause of the phenomena, and not the altered irritability of the nerve. Ziemssen[3] coincides with this view, which he supports by experiments of his own. But he justly condemns the sweeping assertions of Runge, who, forgetting the results obtained in physiological experiments by irritating the nerve with chemical agents, would explain the whole question of electrotonus by a mere antagonism or combination between the polarising and testing currents.

The only experiments on electrotonus in the living man where due care was taken to avoid the innumerable sources of fallacies which beset all such attempts, are those of Cyon,[4] made in 1868. By dint of trouble and care, he obtained normal results in a *few* cases: a significant fact, and which, in itself, speaks volumes against the basing a therapeutical system upon the supposed electrotonic influences of the rough

[1] "Ueber die Polarisation des lebenden Nerven in Menschen," 'Deutsche Klinik,' 1868 and 1871.

[2] "Der Elektrotonus am lebenden Menschen," 'Deutsche Archiv für klinische Medicin' (1870), p. 356.

[3] 'Die Electricität in der Medicin,' 4th Edit., pp. 63 ff.

[4] 'Principes d'Électrothérapie,' Paris, 1873. At the same time I fully agree with all that Erb says of this work in his severe review of it. (Virchow's 'Jahresbericht' for 1873.)

and ready proceedings usual in medical applications of electricity. Cyon experimented on the ulnar nerve; he found it necessary to fix the arm in a plaster of Paris mould; applied the polarising electrodes over points where the nerve was superficial, and tested the extrapolar descending electrotonus by means of induction shocks. The electrotonic condition was expressed in terms of the contractions of the adductor pollicis; their amplitude being registered on a revolving drum by means of a lever attached to the thumb. The cases in which he was unsuccessful he explains partly by morbid conditions of the nerve, partly by the influence of the central organs; but apparently ignores Helmholtz's explanation, which is of far greater importance, that the disturbing element lies in the irregular diffusion of the current from the nerve among the surrounding tissues.

Hitzig[1] in a valuable contribution, unfortunately unfinished, among other points discusses the reactions of the acoustic nerve to the galvanic current. It is the only nerve of the body on which we obtain reaction to anodal opening and cathodal closure *only*. The reason given for this fact by Hitzig is not only satisfactory, but most suggestive. The deviations from the typical formula, he says, occurring in unipolar excitation of other nerves, is due to their being surrounded by good conductors, as suggested by Helmholtz, with reference to Erb's experiments on electrotonus. Now the acoustic nerve is surrounded by bone, a bad conductor, and ends in a substance homogeneous to itself; therefore the whole of it may be thrown into a state of anelectrotonus or katelectrotonus: hence the purity of its reactions. I fully agree with Hitzig in his various strictures on Brenner's views; but think with Erb[2] that it is a fallacy to argue to the absolute non-existence of polar effects from the inefficacy of transverse electrisation of the nerve. May it not be assumed that under such conditions the two opposite polar actions are in equilibrium and neutralise one another?[3]

[1] "Ueber den relativen Werth einiger Elektrisations Methoden," 'Archiv für Psychiatrie,' vol. iv. p. 159 (1874).

[2] "Ueber die Anwendung der Elektricität in der inneren Medicin," 'Volkmann's Vorträge,' No. 46.

[3] The possibility of transverse stimulation of nerves is at present the subject

Filehne[1] has laboured very hard to reconcile the "therapeutical" and "physiological" methods. Considered from a general point of view, such a reconciliation appears unnecessary: impossible indeed from a more special standpoint, since the very essence of the bipolar method consists in acting on the isolated nerve with the two poles at their full undivided power; whilst that of the unipolar method consists in eliminating, as much as possible, by scattering it, the effect of one of the poles. Pflüger having shown that contraction depends on purely polar effects, the problem was rather to explain how it is that with the unipolar method the anodal closure usually preceded the anodal opening contraction, and how anodal closure and cathodal opening contractions occurred at all. Filehne has done this but very imperfectly. He shows that in unipolar excitation the current diffuses both up and down the nerve; and that the results were as if half the neutral pole was placed above, and half below the active pole. We readily grant this, but then are at a loss to understand how he ever could obtain results comparable to those of the bipolar method, as he never opposed but the inferior half of the divided to the whole undivided pole. In fact, his experiments repeated by Burkhardt[2] led this observer to different results. As to his experiments with excessive currents—8–30 cells to a frog's nerve, 50 cells to a rabbit's!—in which he obtained anodal make and kathodal break contractions only, we may safely assume that this phenomenon was rather due to injury to the nerve from the powerful chemical action, and hardly adducible as corresponding to Pflüger's third stage. At any rate Burkhardt did not find it at all constant in its occurrence. It must be noted, by the way, that Filehne does not mention how far he confirmed his data—apparently obtained from very few experiments—by an extended series of controlling tests, a very necessary precaution in this field of inquiry. It has also to be proved that the greater irritability of the upper part of the

of a controversy between Tschiriew, who has affirmed it ('Archiv f. Physiologie,' 1877 and 1879), and Hermann, who denies it ('Handbuch,' 1879, vol. ii. p. 80).

[1] "Die electrotherapeutische und die physiologische Reizmethode," 'Deutsches Archiv für klinische Medicin,' vol. vii., p. 575, 1870.

[2] 'Physiologische Diagnostik der Nerven Krankheiten,' 1875.

exsected frog's nerve is not the result of mutilation, before assuming, as he and others have done, that the anodal stimulation corresponds to the descending, and cathodal to the ascending current. Baierlacher and Meissner, as stated above, held an opposite view.[1] Filehne's views of a "peripolar" stimulation, it may be added, remind one very much of the experiments of Rousseau, Lesure, and Martin Magron,[2] in illustration of the fallacies in the results of Longet and Matteucci, due to the existence of derived currents in the undivided nerve. Chauveau[3] had also shown that the effects of lifting out the nerve on the electrode, and so exciting it by a true "peripolar" current, whilst the other was applied to the limb, were the same as those of unipolar excitation of the nerve *in situ*.

In much of what has been written on the subject of unipolar stimulation, there seems to me to underlie a fundamental fallacy: I mean a confusion between positive and negative *potential*, and positive and negative *pole* (or anode and kathode). It is quietly assumed that when, for instance, an electrode is held in each hand, one of the arms is under an anodal, the other under cathodal influence; but the fact is, they are simply at potentials different, positively and negatively, from that of the earth.[4] It cannot too strongly be dwelt upon that potential means simply *level*, and differences of electrical potential differences of electrical level, without which a flow of electricity is impossible. But by anode and kathode we mean a totally different thing; we mean the point of entrance of the current into, and its point of exit from, an electrolyte, that is, a conductor in which electrolysis is set up. Further, in a circuit composed of several electrolytes, the boundary between each pair of electrolytes acts as kathode to the one which the current leaves, and as anode to the one which it enters.

It is clear then that in sending a current through a composite electrolyte, such as the human body, there can be no question of exclusive localisation of "polar" effects. At every

[1] See Heidenhain, 'Meissner's Bericht,' 1857, p. 420.

[2] 'Gazette Médicale,' 1858; see also Bernard's 'Leçons sur la Physiologie du Système nerveux,' vol. i. p. 180.

[3] Loc. cit. [4] De Watteville, 'Introduction to Medical Electricity,' p. 111.

point where the current passes from one liquid, one tissue, or one cell, into another, there is an anode and a kathode, each endowed with its full chemical and physiological properties. Let us consider what takes place when the positive electrode is applied to the skin over a nerve. The current passes through the epidermis and subjacent layers, and part of it enters the nerve-fibres; at this point we have what I call the *virtual anode.* The greater portion of the electricity which has entered the nerve leaves it almost immediately, on account of the better conductivity of the surrounding tissues: these points of emergence form the *virtual kathode* of the nerve. The relative density of the current at the virtual electrodes depends upon its diffusion; and this again is regulated entirely by physical conditions, viz. the relative position of the actual electrodes, and the relative conductivity of the nerve and surrounding tissues.[1]

Now, it is upon the action of the virtual electrodes that the phenomena of electrotonus, and hence of contraction, depend; and this consideration is, in my opinion, capable of explaining all the apparent anomalies, without any reference to a "peripolar" direction of current, or any such hypothesis. The appearance of katelectrotonus is a more powerful stimulant than the disparition of anelectrotonus. Hence when the positive pole is over the nerve, though the virtual anode is denser[2] than the virtual kathode, the latter will overtake the former, and produce a closer contraction, equal to or stronger than the opening contraction, as soon as $\frac{D^-}{D^+}$ is equal to or greater than $\frac{S^+}{S^-}$ (when D. and S. stand for density and stimulating energy of anode and kathode respectively). Hence also the great preponderance of kathodal closure over kathodal opening: the latter labours under double disadvantage, since the virtual anode now, besides its inherent inferiority, is not sufficiently dense to exert much influence. Brenner, in

[1] If a nerve lie isolated at any point of its course, and placed in contact with an electrode whilst the circuit is closed at any part of the body, the virtual electrodes of the opposite name will be at the points where the nerve emerges from the surrounding tissues.

[2] And hence more effective: Du Bois Reymond's law.

presence of the kathodal opening and anodal closure contractions, tried to explain them away by a mutual "overtaking" (übergreifen) of both poles.

In proportion as we, by the position of the actual electrode, can increase the difference of density between the two virtual electrodes we obtain purer polar effects. It is in this sense that Hitzig's explanation of the acoustic reactions must be taken. With most nerves of the body it is impossible even to approximate the normal formula; but in a few cases (such as with the ulnar at the elbow and wrist, and the peroneal at the head of the fibula) we obtain results which go far to prove my theory. If, for instance, one pole, being as usual on a distant, "indifferent" part of the body (the back, the sternum, &c.), the "differential" electrode is applied over the ulnar, close to the wrist, we obtain a series of contractions, K.C.C., A.O.C., A.C.C. (K.O.C.). Here we remark that A.O.C. > A.C.C. as it should be; but if we push the electrode a little higher up, where the nerve is immediately surrounded by good conductors (muscle and blood-vessels), we find A.O.C. = A.C.C., and finally A.O.C. < A.C.C. The explanation of this fact, according to me, is simply that in the first case the virtual kathode is spread over a greater length of the nerve, hence not so dense and effective; in the second, the current leaving the nerve immediately after it has entered it, the virtual kathode is, though not so dense as the virtual anode, still sufficiently so to make up the difference by its physiological preponderance.

Brenner gives synoptical tables intended to prove the parallelism between the "physiological" and his "polar" method; but with the exception that he shows that "apparition of katelectrotonus" is synonymous with "kathodal closure," and "disparition of anelectrotonus" with "anodal opening," these tables are useless. It would be better to tabulate the different conditions obtaining in the two methods. For instance:—

Physiological or Bipolar.	*Therapeutical or Unipolar.*
The nerve is mutilated and exposed, in direct contact with the electrodes. It is separated from its centres, and from its blood-supply.	The nerve is entire and lies in its physiological and anatomical relations, and is separated from the actual electrode by the skin and other tissues.
The actual and virtual electrodes are one, and the current at each usually of	The current at the virtual electrodes is of different and unknown densities;

equal and known density. They are placed one above the other.

The full electrotonic effects, both stimulating and inhibitory, are produced at each electrode, and the law of contractions is based upon (1) the greater stimulating power of appearing katelectrotonus, (2) the interference of appearing anelectrotonus and of the negative variation of disappearing katelectrotonus.

the whole of one virtual electrode may roughly be said to be between the two halves of the other,

The electrotonic effects are proportional to these densities, and the order and time of contractions depends upon (1) the greater stimulating power of katelectrotonus, (2) the relative density at the virtual electrodes.

In both instances the condition of vitality of the nerve will of course have to be considered as influencing the results. In experiments on the prepared frog's nerve it has long been noted that numerous circumstances, some connected with the process of exsection, others independent from it, exerted a disturbing influence on the results. In many cases these circumstances may be explained on the grounds that wherever the homogeneity of the nerve-fibres is destroyed a secondary anode and kathode exists. Even points where a considerable nerve-branch is given off by a nerve may set up such secondary electrodes, owing to the sudden change in the diameter of the nerve, and perhaps in the relative distribution of the connective tissue, which, of course, is to be looked upon as a different electrolyte from the nerve-fibres.[1] Again, in arguing from unipolar experiments on frogs, a possible source of fallacy may be found in the difference between the relative conductivity of nerve and muscle (and other tissues) in that animal and that obtaining in man: a condition which would materially alter the relative densities of the virtual anodes, under apparently similar circumstances. Any attempt then, I repeat, at *reconciliation* between the unipolar method of the electro-therapeutist and the bipolar method of the physiologist is, strictly speaking, meaningless, for there is nothing to reconcile; they start from the same fundamental axiom. The moment the different conditions under which they act in each case are explained the whole question is as clear as we can wish. In the bipolar method there is an element which does not exist in the unipolar: that of the *inhibitory*[2] effect of the lower pole on the influence of the upper pole. This effect

[1] Hering ('Sitzungsberichte d. K. K. Akad. d. Wissenschaften,' lxxix.) makes similar observations with reference to muscle.

[2] 'Sit venia verbo!'

comes into play only in the third stage of Pflüger's law, where the incipient anelectrotonus arrests the katelectrotonic impulse at the closure of the ascending current, and the negative modification arrests the anelectrotonic stimulation at the opening of the descending current. Trying to find anything similar when the unipolar method is used is a contradiction in terms, a begging of the whole question, and we have seen that Filehne's experiments on the subject are open to grave suspicion.

With reference to the experiments on the production of electrotonus in the human subject, it may be fairly said that they have led to very scanty results, and are exposed to a fundamental objection: that of the summation of the effects of the polarising and testing currents. But do we need any such experiments in order to estimate the electrotonic condition of any nerve? Is not the occurrence of opening and closure contractions, the best proof of the existence of anelectrotonus and of katelectrotonus? Taking the occurrence, then, of these contractions as an index to the condition to the polarised nerve, the obvious conclusion is that it is very rarely indeed, and on a very small scale, that we can ever produce anything like a pure anelectrotonus. In almost every position of the actual positive electrode, we obtain A.C.C > A.O.C.: a proof that the virtual kathode overpowers the virtual anode, that the katelectrotonus predominates over the anelectrotonus. The practical conclusion from this fact is that a therapeutical system built on the opposite anelectrotonic and katelectrotonic effects rest upon an imaginary basis. In by far the great majority of cases we can produce only predominant katelectrotonus; that is katelectrotonus with a more or less considerable admixture of anelectrotonus in its immediate neighbourhood. When to this consideration we add the fact that anelectrotonus immediately passes, on breaking the current, into a phase of increased irritability (positive modification) it is difficult to understand the precepts so often given that "the positive pole acts as a sedative, the negative as a stimulant." Both are stimulants, if "stimulation" there be, the kathode more so than the anode; but that is all, and I demur to the charge of inconsistency brought by Erb against those who,

adopting the unipolar method for purposes of diagnosis, do not carry it out systematically in therapeutics. A further objection to the electrotonic system of electro-therapeutics is that in the only instance where we can produce pure polar effects, in that of the acoustic nerve, "the systematic production of an- or katelectrotonus," as it is called, has not proved a success. Indeed, if the doctrine were true, we might expect that where the negative pole is indicated the positive would increase the mischief, and *vice versa*. It is a matter of daily experience that hyperæsthesic, hyperalgesic, and hypercinesic symptoms are, as a rule, happily influenced by the one pole as by the other. As to the pretension of inducing electrotonus of the brain, spinal cord, and vaso-motor nerves (katelectrotonus of the latter, by the way, to produce *dilatation* of the arterioles), I do not know upon what physiological evidence it rests, nor even what the expression very well means.

Though the unipolar method does not fulfil therapeutically the ambition of its promoters, its adoption has led to most valuable results in the field of diagnosis, and I am the more anxious to recognise Brenner's durable services in this respect that I have been led to stand in antagonism to his other views. Electro-diagnosis, difficult enough on this unipolar system, is impossible on the bipolar method, with which in the living subject we would get *four* electrodes to the same nerve; and the bitterest foes of the new system have been obliged to adopt it, though not always with the best grace.[1] A clear conception of the physical conditions of unipolar stimulation is, however, necessary for the rational application of electrical tests, and I am not without hope that the previous remarks may clear up certain obscurities hitherto prevalent.[2]

The question of a possible influence of the direction of the current is intimately bound up with that of the density of the current. On this point, again, much useless discussion has been expended. Supposing it is desired to estimate how much electricity passes through the sciatic nerve at a point half

[1] Cf. Benedikt, 'Elektrotherapie,' 2nd Ed. 1874.

[2] When the electrode is placed on the abductor indicis near its insertion, I find that A.C.C. > K.C.C. The same phenomenon occurs in other parts, and is explainable on the assumption that a virtual kathode occurs at a point of the muscle more excitable than the point of application of the anode.

way between the glutæal region to the popliteal space when the electrodes with a current of say twenty millevebers [1] are placed in these positions, all we shall have to do is to divide the diameter × specific resistance of the nerve by the diameter × mean specific resistance of the tissues of the thigh. The equation $\frac{D \times R,}{D^1 \times R^1} = \frac{X}{20}$ gives us the result. Assuming $D^1 = 12$ and $R^1 = 5$, when D and R are taken at unity this would give us $\frac{1}{60}$th of the current as passing through the nerve at the point, which is a very high estimate. This rough calculation gives us at least as accurate quantitative results as the galvanometric experiments of Burkhardt [2] and others, since the unknown relation between the resistance of the piece of nerve included between the needles and that of the galvanometer used can hardly be determined. This is not the place to discuss the arguments adduced by those who uphold the influence of the direction of the currents on the various tissues.[3] They cannot escape the objections deduced from the fact of a peripolar direction of the current in the nerve and from the great diffusion of the current in the interpolar region; it is only within a short radius of each electrode that the density is sufficiently great to produce physiological effects. And though the therapeutical conditions drawn from the electrotonic influence of the poles are not justified, it has yet to be shown that the very weak currents which traverse the tissues at a distance from the electrodes have any curative power, apart from any demonstrable physiological action. This supposition is, of course, within the range of possibility; but the partisans of direction-influence have hitherto produced no proof that their results do not depend from the relative position of the poles. The discussion of this point will be best deferred to another occasion.

If these pages, as I hope, have shaken one of the many *Idola Specûs* of electro-therapeutics, and disposed of some of the loose talk and thought so rife in the literature of the subject, my object is fulfilled.

[1] De Watteville, 'Practical Introduction to Medical Electricity,' chap. i. A current of 20 millivebers would be given here by about 40 Daniells.

[2] "Die polare Methode," 'Deutsches Archiv,' 1870.

[3] Onimus and Legros, 'Traité d'Électricité médicale,' Paris, 1872.

ANATOMY AND PHYSIOLOGY OF THE CHORDA TYMPANI NERVE.

BY HORATIO R. BIGELOW, M.D., WASHINGTON, U.S.A.

A SERIES of experiments, extending over a number of months, may, perhaps, give me a right to be heard upon the question of the "Anatomy and Physiology of the Chorda Tympani." It was not without much diffidence and a measure of embarrassment that I published, in June, 1879, in the 'Archives of Medicine' (New York), the conviction which I had reached that the chorda tympani is not a branch of the facial proper, but the continuation of the intermediary nerve of Weisberg, and that its physiology differed essentially from that claimed for it by writers upon special subjects of scientific medicine. I felt that I stood alone in a large field already worn by the onward march of physiologists, and that while their footprints all tended in the same direction, mine were bending in a line entirely opposite. But now that Dr. E. C. Spitzka ('New York Medical Record,' Jan. 31st, 1880) confirms my views in greater part (his objection to my theory of the function of the ganglion will be noticed farther on), I feel that a perfect vindication of such discoveries is merely a question of time. Prof. Sappey ('Traité d'Anatomie Descriptive'), than whom few write more clearly and forcibly, and whose opinion will always command profound consideration, writes: "La dissection unie à l'emploi des réactifs démontre entre la corde du tympan et le lingual une fusion intime, complète *fibrille à fibrille* dans toute l'étendue de l'adossement de ces nerfs; à l'aide de ce procédé on tenterait donc vainement de reconnaître le mode de terminaison de la corde du tympan." It

was this view of its anatomy which first led me to inquire most thoroughly and honestly into the relations of the chorda tympani, and, while my deductions are opposed in every particular to those of Prof. Sappey and of neuro-physiologists, yet may they be kindly received, and substantiated by individual investigation. In the 'New York Medical Record' for Jan. 17th, 1880, I published the following conclusions:—

1. The chorda tympani is distinct and integral throughout its entire length.

2. It is derived from the nerve of Weisberg, and not from the facial.

3. Its especial sensory function is derived from the ganglion upon the nerve of Weisberg, into the granular protoplasm of which the ultimate fibrils may be traced.

4. The lingual branch of the fifth presides over general sensibility only. Isolation of the chorda tympani destroys the sense of taste in the anterior two-thirds of the tongue, the fibres undergoing degeneration.

5. Section of the lingual destroys sensibility, but only modifies the sense of taste; this modification being due exclusively to the branches from the chorda tympani.

6. Section of the facial, behind the origin of the chorda tympani, destroys the sense of taste *only after a lapse of time;* and this not because the facial at this point contains gustatory filaments, but because the nerve is cut off suddenly from its supply, and has received such a shock that it undergoes degeneration. If the chorda tympani be drawn out at the point where we first notice its filaments of origin, and divided, the sense of taste will be almost entirely destroyed. If the nerve of Weisberg be cut in the aqueduct behind the ganglion, the sense of taste is lost. From which it may be inferred that the intermediary nerve is continued in the chorda tympani, and that this latter is a carrier of the sense of taste from the cells in the intumescentia gangliformis.

The differentiation of the intermediary nerve and the main trunk is attended with the utmost difficulty, and can be effected only by the use of reagents, "teasing" all the fibrils under a good lens mounted upon a tripod. In this way the chorda tympani may be separated from the lingual and followed

up: the terminal filaments expanding into the taste cells of the beakers, the bulbous expansion at the end being identical with the transparent nuclei. In the same way, by using a power of 1000 diam., we may trace into the intumescentia gangliformis the nerve fibrils of the intermediary nerve in the processes of the ganglion at their junction with the cell, and these may be traced into the granular protoplasm. From Dr. Spitzka's note in the 'New York Medical Record' of January 31st, I may be allowed, for purposes of discussion, a liberal quotation:—

"I am able to add an important confirmatory observation to those published by Dr. Horatio R. Bigelow in the 'Record,' favouring the view that the chorda tympani nerve is, after all, not a branch of the facial proper, but the continuation of the *nervus intermedius* of Weisberg. It is well known that this latter nerve is entirely distinct from the facial at the origin from the medulla oblongata. In numerous transverse microscopic sections of human and animal peduncular tracts, I have found that the fibres of the nervus intermedius have *no* connection with the facial nerve nuclei. Their central termination lies, in fact, in lower altitudes; that is to say, while the facial nuclei are situated within the lower pons margin, the nucleus of the nerve of Weisberg lies strictly within the limits of the medulla oblongata in the level of the superior auditory nucleus. The gray origin of the nerve of Weisberg is diffuse, and, in the *strict* sense of the term, cannot be called a nucleus for that reason. On examining its relations more closely, we find that it *does not correspond to the motor gray column* of the medulla, but to an ideal continuation of the gelatinous column of the trigeminus region; in other words, it originates in the *sensory* gray column of the medulla. . . . . Dr. Bigelow makes one statement to which I must take exception. He attributes the sensory function of Weisberg's nerve to a ganglion on the nerve. It is generally conceded that peripheral ganglia are obscure in their function, and that the function of a cerebro-spinal nerve is determined by its *central* connections."

This confirmation, in part at least, of my own views was most opportune. It was gratifying to be sustained in so

important a matter by one so skilled in investigation as Dr. Spitzka is known to be. Unquestionably it is a fact that the consensus of medical opinion concedes that the function of a cerebro-spinal nerve is determined by its *central* connections, rather than by peripheral ganglia. But such concession is due to the seduction of pleasant theorizing rather than to absolute demonstration. No one has attempted a logical explanation of the functions of the peripheral ganglia, and their existence upon sensory nerves seems to be ignored in the discussion of the characteristic nerve function. It may or may not be true that the functions of *all* cerebro-spinal nerves are determined by a *central* connection; we have no demonstrable proof for or against the theory. In all exact physiological inquiries, as, indeed, in all inquiries of exact science, we must be governed by fact rather than by inference. I doubt exceedingly if the chorda tympani is made sensory solely through its central connection. I can make no absolute assertion, because experiments upon this ganglion are surrounded with almost insurmountable difficulty. I have exposed it, and touched it with acetic acid; but a microscopic section showed that the chemical action had extended along the nerve fibre, and that it had lost its normal characteristics. I then tied the nerve with a small silk thread behind the ganglion, and irritated the ganglion with the electrode of a small Gaiffe battery; the sense of taste persisted for two hours and a half, so that there must have been reserve force at least within the ganglion. If it were possible either to extirpate the ganglion, or to destroy it without resultant injury to nerve fibre, the question might be definitely settled; and to this end I have called to its fullest extent upon the little inventive genius I may possess, but so far unavailingly. I do not maintain that the intumescentia gangliformis in question is capable of originating the sensory function, although in some cases of paralysis it would seem to possess such a power; but one of my experiments has demonstrated that it does act either as a storehouse or as a generator. The ganglion, too, serves to differentiate with exquisite precision between the effects of sapid substances, whose appreciation is governed by a central connection; the psychic phenomena of the cord

contrasting strongly with the physical manifestation of the ganglion.

In closing this paper, I may be allowed to refer once more to my communication in the 'Record.' "The difficulty of noting the impairment of taste is exceedingly great, and one will fail often before arriving at satisfactory results. The shock following a section of the nerve within the cranial cavity necessarily modifies exact conclusions, and, as Flint very justly observes ('Text-Book of Human Physiology,' p. 761), 'We must remember the difficulty of such observations, and we are to rely mainly upon the unmistakable phenomena noted in cases of affection of the chorda tympani in the human subject.' Paralysis of the facial, behind the origin of the chorda tympani, is attended with loss of taste in the anterior two-thirds of the tongue. But this is not due, as I believe, to the existence of gustatory filaments in the facial itself, but to the fact that the transmitted shock is so great as to destroy the function of the ganglion upon the nerve of Weisberg, and in this way inhibit the special characteristic of the chorda tympani as a carrier of the sense of taste. The sensibility of the tongue remains, because it derives its sensory power from a different source; the cells furnishing the lingual branch of the fifth are not interfered with in such cases of paralysis. It is a fact, however, that sensibility is often modified, owing to the inability of the nerve fibre, by reason of a degeneration resultant upon shock, to carry the sensation. If we isolate, so far as it may be possible to do, with a proper regard for the life of the animal, both the chorda tympani and the intermediary nerves, the sense of taste becomes greatly modified, and after a time almost entirely lost, the feeble sense remaining being due to the fibrils which it is impossible to divide. If there were essential gustatory filaments in the facial, the sense of taste would persist in a greater degree than it does after division of these two nerves. If we are to study the functions of the chorda tympani from its action in those morbid conditions affecting it, the nature of the *shock*, as well as the resultant influence engendered by contiguity of structure or by actual contact, must never be lost sight of. Nerves, themselves uninjured, may suffer in their action by transmitted influences."

ON THE PARALYSIS WHICH RESULTS FROM ANGULAR CURVATURE OF THE SPINE.

BY JOHN DUNCAN, M.D.,

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CASES of caries may be divided into two classes, of which the types are very distinct, although they shade into one another with an infinite gradation. In the one class are those in which the disease has a constitutional, in the other those in which it has an external etiology.

In the spine it appears to me that this distinction is as distinctly marked as in other parts of the body. Patients of the first group have the strumous or tubercular diathesis; near relations have had phthisis, or white swelling, or glandular inflammations. They have themselves the general characters of the diathesis, or have suffered from its pathological manifestations. They have spat blood or had pleurisy. They have scars in the neck, or tubercle in the lungs, or chronic joint-disease. In short, the personal or family history is bad.

In such cases the angular curvature is rapid in its formation. Psoas, or lumbar, or cervical abscesses form early in the disease. The patients sink from hectic or from amyloid degeneration of the liver and kidney. If paralysis make its appearance it is commonly from pressure on the cord, and is sometimes suddenly fatal in the cervical region.

Patients in the second class have generally some distinct history of serious injury. Their appearance is robust and healthy. The family and personal history is free from trace of strumous disease. Abscess is comparatively a rare occur-

rence. Paralysis, on the contrary, appears soon. Sometimes it precedes the projection of the spines, and is accompanied only by tenderness on pressure or percussion, or on the application of heat or cold. The patients die from the effects of the paralysis, and if there be no paralysis the disease runs an exceedingly slow course. While the strumous form attacks the child or the young adult, this variety is more common in middle or advanced life.

The paralysis is peculiar. It confines itself specially to the motor track of the cord. Sensation is rarely affected. The motor paralysis is often of the spastic form, and is sometimes very extreme. The lower limbs are most apt to become paralysed. The bladder and bowels retain their normal functions, or are only temporarily disturbed. Trophic changes also are infrequent.

I have been much struck by the distinct way in which all these peculiarities come out in the series of cases which I shortly append. They are those which have been resident in the wards under my care during the last two years, having been admitted for the purpose of opening the abscess or for observation. Many other cases of the strumous variety were out-patients during the same period, but I have not met with any other examples of paralysis.

I.—Jessie S., æt. 31, married, admitted 18th December, 1878. Sallow and thin. Her mother died of phthisis, and her father died young. She is very subject to indigestion. About 4 years ago she felt pain in the back after delivery of her second child. She was unable to carry it about, and has not been free from the pain since that time. Three months ago swelling appeared about Poupart's ligament, which was opened. There is prominence of the 11th and 12th dorsal vertebræ. A plaister jacket was applied. In June, 1879, the discharge, though slight, still continued, and there was little alteration in the general condition.

II.—Harriet M., æt. 23, single, admitted 19th June, 1878.

History. *Family:* Mother and two sisters died of phthisis. A brother had an abscess in his side, which has now healed.

Personal: Amputation of the third toe of the left foot was

performed a year ago, on account of caries. There is now caries of the metacarpo-phalangeal joint of the left thumb.

There is lumbar abscess, and projection, and pain on pressure over the 4th and 5th dorsal vertebræ.

The abscess was aspirated and a jacket applied. When last heard of, the patient was progressing favourably.

It would be tedious to enter into the history of common cases of which these may be taken as the types. In all the treatment was either aspiration or antiseptic opening of the abscess, with the use of the plaister jacket. I enumerate only and mention the salient points.

III.—Duncan C., æt. 22. Psoas abscess and angular projection of the 10th and 11th dorsal vertebræ. Has twice had inflammation of the lungs, and the present signs of phthisis are distinct. A brother had white swelling of the knee.

IV.—John L., æt. 23, admitted 7th November, 1879. Psoas abscess and angular curvature of 6th, 7th, and 8th dorsal vertebræ. He refers his disease to a fall downstairs, but it is clearly ascertained that the projection existed before the fall, although the pain was thereby increased. Some years ago he had abscesses in the right arm, which lasted several months. His mother died after child-birth.

V.—A. D., æt. 3, admitted January, 1880. Double psoas abscess and excessive curvature in lower dorsal region. Two cases of phthisis in the family. The treatment is by double extension.

VI.—Patrick D., æt. 31. Pale, thin, and strumous-looking. Lumbar abscess and angular curvature of 7th dorsal spine. Knows nothing of family history. No injury or previous illness.

VII.—William S., æt. 25. Lumbar abscess and projection of 7th and 8th dorsal vertebræ. No history of injury. A brother has scars in his neck.

VIII.—John N., æt. 15. Caries of 3rd and 4th cervical vertebræ. Father died of phthisis. In this case I opened a retro-pharyngeal abscess by the method advocated by Mr. Hilton and Mr. Chiene. Unfortunately it opened also into the pharynx a week afterwards, and consequently became septic. He comes in to show himself occasionally, and is

getting much emaciated from the discharge, which has now been going on for more than a year. He has also had several fits. The epilepsy, however, I believe to be connected with carie-necrosis of the right temporal bone in the mastoid region, which is considerable in extent.

In none of these cases was there the slightest paralytic symptom.

The following cases illustrate the other form of disease of the vertebral bodies to which I have referred.

I.—The first case which attracted my notice was that of a miner, æt. 23, whom Professor Saunders asked me to see in his ward. There was marked angular curvature of the lowest cervical and upper dorsal vertebræ, with an unusual amount of inclination to the left. He said he had been in the habit of carrying exceedingly heavy weights on the left shoulder, and on several occasions was conscious of having strained the parts. In fact, he felt assured that he had immediately produced the lump in one of his "lifts." There was considerable pain on pressure. He had spastic paralysis of the lower extremity to an extreme degree. The tendon reflex was enormously exaggerated, and he was unable to walk more than a few paces, even with assistance, on account of the spasmodic jerking of his limbs. I applied the plaister jacket, with jury-mast and elastic support for the head. He at once began to improve. He still after a year continues to wear the apparatus, and sends me word that he can go about as well as ever, but his legs, he thinks, "make him walk faster than he would otherwise do."

II.—John K., æt. 27. Admitted December 7th, 1878. Family and personal history good.

A year before admission he fell from a ladder while carrying a heavy load of lead. He injured his back, and thereafter wore a belt for two months before he observed the projection of his spine. The 5th, 6th, and 7th dorsal vertebræ are prominent. He has distinct loss of power in the legs, which he moves in a jerking manner. The patellar tendon reflex is increased. Sensation is normal. He had a plaister of Paris jacket applied. The pain and paralysis much improved when he was last heard of, six months after leaving. He then showed himself for renewal of the jacket.

III.—John D., miner, æt. 53. Admitted May 6th, 1879. The family and personal history are unexceptionable. He is not aware of any special injury, but says that he has often felt his occupation a strain on his back.

He has felt pain in his back for about a year, and noticed the projection of the 4th dorsal spine several months ago. For three or four weeks his legs have been getting weaker. He is now barely able to walk with assistance, and his legs have a great tendency to cross in doing so. The tendon reflex is much increased in every action. Sensation is slightly duller than normal in the lower limbs. The bowels are sluggish. A jacket was applied, but fitted badly, and during the ten days he was in the infirmary he rapidly deteriorated. At the end of that time he was quite unable to stand alone. A better jacket was adjusted, and he at once began to improve. He returned January, 1880, to ask whether he might resume light work. He could then walk without the least apparent impediment.

IV.—Henry B., æt. 13. The family history is good. There is no history of injury. The 3rd, 4th, and 5th dorsal spines project. The disease has been in progress for three years, but there is no sign of abscess. On the other hand, the spastic paralysis is so marked that he cannot walk even with assistance. He has had a jacket on now several months, but although the local pain is relieved the paralysis is steadily getting worse.

V.—Thomas B., miner, æt. 32. Admitted 6th Feb. 1880, a robust-looking man; family history perfectly good.

As a boy he pushed waggons in the mine by laying his head against them, and remembers straining his neck when he was seventeen so severely that the pain lasted for a year, during which he could not work. Since then the neck has been stiff. Six years ago he lost the power of his fingers, and shortly afterwards of his legs. The fingers are now nearly well. The legs have got gradually worse, and he is now quite unable to walk. He has on several occasions had difficulty in passing water. This was much relieved by the application of a blister to the nape of the neck. The actual cautery has been used three times, twice with decided but temporary

improvement in the paralysis. Sensation is not affected; he can move his limbs pretty freely in bed, but cannot support himself. Tendon reflex is increased. The neck is shortened, and quite stiff. There is no abscess. The plaister jacket was applied 9th Feb. 1880.

VI.—William B., miner, æt. 38. Admitted July, 1879. Family history good. No previous illness. He once broke his thigh, and has had several severe injuries to the back, but cannot attribute his present illness to any one in particular. He has had pain in the back for two years, but left off work only five weeks ago, when the doctor observed the prominence of the 4th dorsal spine. The patient walks with great difficulty, and has all the symptoms of spastic paralysis. The leg continues to twitch for a considerable time after the stroke on the tendon. A plaister jacket was applied, when he immediately began to improve. It was renewed in Jan. 1880, when the paralysis was found to have almost disappeared.

VII.—James McGlore, æt. 37, miner. Patient of Dr. Muirhead's. The family history is unexceptionable. Four years ago, just before his illness commenced, he slept out all night while intoxicated. For two years he had paralysis and pain in the back, but no projection. The 3rd dorsal spine is now prominent. The legs are constantly and rigidly contracted, flexed, and crossed. If the great toe be forcibly flexed the limb is involuntarily straightened, but soon recovers its abnormal posture. Two years ago he had some difficulty in urination and defæcation, but it speedily passed off. Double extension was used, and the actual cautery applied, but without benefit.

VIII.—W. M., æt. 38. Admitted Feb. 1880. The family and personal history is good. He has met with no serious injury. Pain in the back came on about six months ago, with projection of 12th dorsal spine. He has distinct spastic paralysis with increased tendon reflex. There is no abscess. A plaister jacket has been applied.

The following cases differ from the others in several important features, and may be regarded as links in the chain which unites the two classes together:—

I.—James H., æt. 42. Patient of Dr. Muirhead's. A brother

died of phthisis. The disease began four years ago, without known cause. Pain in and projection of the 3rd, 4th, and 5th dorsal spines were at once observed, and an abscess soon afterwards formed a little lower down, which still continues to discharge. In August, 1879, he began to feel loss of power in the limbs, and abdominal constriction. He has now a trailing gait. The muscles are flaccid, and without spasm. The reflex action is diminished. The patellar tendon reflex is slightly increased, especially on the left side.

II.—Patrick L., æt. 38, labourer. Admitted July, 1879. Family history good. He met with a severe injury to the back by falling from a ladder a year ago, and immediately afterwards a gradually increasing projection of the 5th dorsal spine commenced, followed shortly by an abscess a little lower down. There is no paralysis. The abscess was opened antiseptically, and he is now progressing favourably.

These cases, I think, mark very clearly the distinction I have drawn between the strumous form of the disease and that which has its most striking analogy in the chronic interstitial absorption of the neck of the thigh-bone, which in adults so often follows severe injury. It will be noted that in this latter variety, to which I specially wish to direct attention, there was in almost every case a distinct history of injury, or of such an occupation as throws great strain upon the vertebral column. In the strumous the disease may have its starting-point in an injury, but most often the history is an afterthought, or the projection is said to have come of itself. The other peculiarities of caries siccum, to which I have already referred, are also remarkably illustrated by these cases. Of all these features the most singular seems to me the frequency with which it is accompanied by paralysis. I have no doubt that my experience of these two years has been unusual. Dr. Saunders's patient, an Ayrshire miner, was so greatly benefited by treatment that the patients T. B. and J. D., also miners in the same district, were induced to come by the report of his case. Still it is plainly much more frequent in this than in the strumous caries.

It will be noted that the paralysis was almost exclusively motor. It is a feature of all spinal disease that special tracts

of the cord should be very accurately marked out by it. Whatever the explanation, it appears to characterise this consecutive paralysis as distinctly as the idiopathic. One can, to a certain extent, understand how the motor portions of the cord are most prone to become inflamed in consequence of caries of the bone. But it is more difficult to determine why the spastic form of paralysis should so greatly preponderate. In Dr. Saunders and Dr. Muirhead's patients the disease was severe and typical; in others it was unmistakable, though not so far advanced. The cases are clear examples of those secondary degenerations which Türck and Charcot have described.

The success of the treatment has been gratifying. Five cases have been sufficiently long under treatment to allow of a judgment being formed. Three of these have been practically cured, although they still wear the apparatus. In one case no benefit has resulted from the jacket. In another the jacket was not applied as it had previously been tried in Glasgow. For him I used the actual cautery without effect, and permanent extension in bed from head and feet, was applied, but not persisted in from the annoyance and inconvenience it occasioned.

It is plain that in this chronic ostitis, as in the same disease in other parts of the body, our local treatment is twofold, rest and counter-irritation. These cases illustrate the advantage that may be gained from both. The miner, Thomas B., was benefited on many occasions by blisters, and twice by actual cautery. Unfortunately the relief was not permanent. I fear that if counter-irritation be used alone this is the usual result, whether blisters, seton, or cautery be the means employed. To do much good they require persistence, and with persistence comes a time when their beneficial effect ceases. They must be combined with rest. Now rest for the spinal column is difficult to procure. It implies abrogation both of motion and of pressure. This may be attempted in two ways; by retention in bed, or by the use of apparatus. Until Mr. Sayre made known the value of the plaister jacket, the inefficiency of apparatus was such that recumbency was the recognised treatment for disease of the spinal column, at least in the

Edinburgh school. But if the objects aimed at by apparatus be attained, its other advantages are manifest; and I believe that they are in great measure so attained by the jacket. It does not take off perpendicular pressure so completely as recumbency does. If the jacket alone be used, the body telescopes gradually into it, and the plaister sinks *en masse* unless it be very accurately adjusted to the pelvis. If the disease be at or above the 6th dorsal vertebra, it is essential that the jury-mast be also applied, and the head attached to it by elastic bands which act much better than rigid straps. But while apparatus may not thoroughly relieve pressure, at least it very effectively restrains motion, which mere recumbency cannot. Even the actions of breathing are diverted from the ribs.

It is not essential that the material be plaister of Paris. That is usually most convenient. But I have moulded very comfortable jackets from the poroplastic splint material, and paraffin is light and easily applied in children, though not so durable and rigid in the adult.

The conclusions to be drawn from these cases are:

1st. That there are two distinct varieties of inflammation which attack the bodies of the vertebræ.

2nd. That in strumous cases there is comparatively little tendency to affection of the spinal cord.

3rd. That in chronic interstitial absorption there is a great tendency to paralysis, which presents the usual characters of what has been termed "pressure myelitis," with its secondary degenerations.

4th. That this paralysis may often be cured by rest and counter-irritation.

ON SOME VARIETIES OF CERVICAL PARAPLEGIA.

BY THOMAS BUZZARD, M.D.,

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It has happened to me to have under observation, more or less recently, several cases of the condition to which the term "cervical paraplegia" has been (first of all, I think, by Sir William Gull) applied. In these cases paralysis, usually both of motion and sensation, has affected more or less completely all four extremities, the upper in excess, and the circumstances by which the paralysis has been characterised have enabled one without difficulty to localise the lesion in the upper part of the spinal cord. In only one case, however, did the result give the opportunity of confirming the diagnosis by a post-mortem examination, and on this account I propose to relate this example first. The symptoms during life, and the pathological appearances after death, will serve to throw light upon many points in the other cases described, which have terminated in a greater or less amount of recovery. In spite of the aid thus afforded, it will be found that although the locality of the lesion can usually be indicated with tolerable confidence, its nature is by no means always to be determined with equal certainty.

Case I.—*Epileptic seizures—incomplete paralysis of arms and legs; pseudo-neuralgic pains; amyotrophy; marked improvement under iodide and mercury; relapse; apoplectic seizure; death.* Autopsy:—*hypertrophic cervical pachymeningitis; transverse myelitis; thickening of basilar artery; thrombosis; cerebral softening.*

The patient, S——, was a clerk, about 35 years of age, living a few miles from town, whom I visited at the request of his employer, on July 14th, 1877. He was below the middle

stature, with a squint, which I was told was of many years standing. He presented a peculiarly muddled look, and a manner so confused that I had difficulty in obtaining information from him. His memory appeared to be very bad. From what I could gather then, supplemented by later information, his family history was as follows. His father died at 38 years of age, in a lunatic asylum; his mother, æt. 50, of a "natural death;" she drank. He had never had any brothers or sisters.

Personal history.—The patient suffered from fits in his infancy until he was about six or seven years old. The next thing one learnt was that when he was 21 years of age he lost the sight of each eye in succession, that of the left eventually recovering. About eight or nine years ago he had a venereal ulcer, which was accompanied by two suppurating buboes, for which he took medicine to make his gums sore. His employer tells me that five or six years ago S——'s eyes were "bad" (interstitial keratitis?), so that he had to be off work for six months. During the last year he had had five or six fits, of which two occurred in the week preceding my visit. On the occasion of the first of these he felt dizziness, and was obliged to lay hold of his desk. He thought he was going to fall, and shortly afterwards he did fall, unconscious. He is not aware of having bitten his tongue.

Present state.—The sight of the right eye is almost nil, on account of a large leukoma of the cornea, concealing nearly the entire pupil. The squint, which is said to be of old standing, is not accompanied by double vision. He walks feebly and with a tottering gait. There is no power of grasping with either hand. This failure of strength, he says, has only taken place during the last four days, but for the last week he has been feeling increasingly feeble. There is no marked want of symmetry in the face. His tongue is protruded straight. The patient cries out every now and then with aching pains in the arms, sometimes in one and sometimes in the other. Frequently there is much pain over the brows and across the back. At times, he says, he suffers from pain all round the lower part of the head behind.

For some months past he has been liable to what he calls "rheumatism" in his limbs, and aching pains across the head

and loins. There is what he describes as "painful numbness" in both arms.

The heart and lungs are healthy. Appetite and digestion bad.

By the ophthalmoscope (under great difficulty, owing to unsteadiness, very small pupil, and some considerable haziness of the media) the retinal vessels in the left eye appeared tortuous, and the outline of the disc indistinct. The large opacity precluded observation of the right fundus oculi.

His landlady told me that six or seven weeks before this his manner had appeared very strange, and his memory had failed him to a large extent. It seemed that for a week past he had been taking small doses of bromide. I ordered perchloride of mercury and iodide of potassium.

A week later he came to my house, distinctly improved in intelligence. I noted that he held his head as though he had a stiff neck, and complained of pains at the back of the head and across the right ear.

There was a peculiarly helpless look about his hands. The excitability of the muscles of his arms was diminished in a marked degree to the faradic current.

Whilst examining him with the ophthalmoscope, I noticed that he was constantly swaying about, and frequently exclaiming suddenly as from the effects of pain. His arms would jerk, and he complained of sharp pains in them.

His temperature was normal. The urine contained no albumen or sugar.

After another fortnight's treatment with mercury and iodide, he was manifestly much improved. There had been no repetition of a fit. He had lost the pains in his arms, and there was but little pain in the head. The right leg was stronger. He described a feeling of tightness around the waist, which he had experienced for two or three days, but which was relieved by the action of the bowels. The mind appeared to be quite clear. By the ophthalmoscope the left disc appeared to be more distinctly defined, but the vessels remained tortuous. He was ordered one-sixteenth of a grain of perchloride of mercury with twenty grains of iodide of potassium three times a day, and twenty grains of bromide of potassium at bedtime.

On August 3rd the following note was made: "Three days ago he had a repetition of a feeling which he had not experienced for a year previously, and which then ushered in a fit. The feeling was of a whirling round in the centre of his head. He had a trifling amount of pain at the back of the head.

"His walking power improves daily. He walked to-day the best part of a mile, although there is still weakness, especially in the right leg.

"There is wasting of the dorsal interossei in each hand, but the fingers have regained warmth and colour (they had been cold and dead-looking). Their movements are, however, still very clumsy. This morning he lifted the ewer, which he could not have done three days ago.

"The electrical reaction in the muscles of the forearm is somewhat improved."

The following note of diagnosis was also made at this time: "One would think, from the powerlessness and wasting of the muscles, together with loss of electric irritability, that here was a case of pressure upon the anterior roots of nerves, probably from pachymeningitis."

Three weeks later the patient had gained strength; and was able to walk as much as three miles. The arms, too, had greatly improved. He had resumed his usual handwriting with a pencil, but there was too much clumsiness in the fingers for the successful use of a pen; nor could he pick up coins or pins. "It was only the fingers," he said, "which were still obstinate and had not regained their pliancy." The hands felt generally warm in the evening. The warmth of the bed seemed to soothe them. Contact with metal appeared to send them "all ajar." There was no pain in the head: his appetite was good and he slept well. He had been taking the iodide with a little iron.

On September 12 it is noted that the patient can walk three to four miles without fatigue. The grasp of the left hand is very imperfect, and there is still wasting of the interossei, as well as an "electrical sensation" in that hand. His gait is rather jerky and disorderly. He is somewhat restless at night. There is a sense of fulness, but no pain, at the back of the head. A few days after this he began to get more pain in the right arm,

especially from the middle of the upper arm down to the tip of the ring-finger. On examination, the ring and little fingers of the right hand were found to be clumsy, and unnatural in their movements.

September 20th.—There has only been a little pain, "almost what he might describe as a sympathetic feeling," in the left arm. But the fingers of the left hand are much more helpless and clumsy than those of the right.

"At this moment" (I note) "there is no pain in the right arm, but he has had several paroxysms this morning and many in the night. The pain is almost unbearable. It comes on very suddenly—a burning aching pain, 'as if the vein were filled with molten lead.' The day before yesterday the pain was so intense that he 'started up, and broke into a perspiration all over.' A paroxysm will last about half a minute. It seems to begin at the top of the arm, and it 'must go its course.' After the pain has left, a feeling of relief alone remains. He can lift his arm during the pain; in fact the exertion of the muscles seems to give relief. It is usually when the pain is going on in the right arm that he gets a little pain in the left. But he thinks (questioned) that he has sometimes a little pain in the left arm when none is going on in the right."

Examination showed that the excitability of the muscles of both arms to induced currents was much diminished, but not removed. There was still wasting of the interossei, but not, it was thought, so great as had been. He was unsteady on his legs, especially on the left.

A week ago he felt pretty well, and went by rail to the Crystal Palace. He undressed in my room for examination, and appeared to be very helpless; I had to button his clothes. By the ophthalmoscope, I found the retinal vessels of the left fundus very tortuous, but the outline of the disc distinct enough. I dilated the right pupil with atropine, but the opacity prevented any observation of the fundus. Three days ago he went and dined with a friend in the City (a distance of some six or seven miles). The following day was spent indoors.

The patient was now placed under the care of Mr. Harrison, of Streatham, whom I met in consultation on November 1.

S—— was up and dressed, seated in a chair. The arms were much atrophied, and the legs also, but to a less extent. He did not complain of pain in the arms, but occasionally I observed them to be jerked, and then he would cry out. When asked to explain the cause, he said that he had the cramp in them.

The left forearm showed hyperæsthesia to cold, but there was delay in receiving the impression of pinches.

There was paralysis of the sphincters, with ammoniacal urine.

The patient was put on a water-bed and ordered one-sixteenth of a grain of perchloride of mercury with fifteen grains of iodide of potassium three times daily; also some lactophosphate of iron.

November 17*th*.—I heard that there was complete paralysis of the intercostal muscles, and the breathing was entirely diaphragmatic. The patient was having epileptiform attacks every day. He was thought to be dying.

November 20*th*.—As the gums were beginning to be sore, the mercury was stopped and the iodide continued.

December 10*th*.—Mr. Harrison wrote that there had been a slight improvement. The intercostal muscles were acting slightly, and there was more power in the arms and legs.

About a week before his death, which took place on December 24th, he was removed into other lodgings. At this time he could just slowly and tremulously lift a handkerchief and give a very slight squeeze with either hand. The arms appeared to be equally weak.

For many weeks before the end he could not stand, and was indeed unable to lift his foot from the floor; but during the last few days of his life he could do this. Towards the close he became exceedingly irritable in temper.

On December 23rd, he was taken with insensibility and stertorous breathing, and died the following day, having never recovered consciousness.

The autopsy took place 52 hours after death, in the presence of Mr. Harrison and myself. Weather very cold. The body was extremely exsanguineous; a small bedsore on the sacrum. Much wasting of muscles was noted in the forearms and hands, not so much in the upper arms. The legs were thin, but did not appear so much atrophied as the upper extremities.

Head.—There was some slight opacity of the arachnoid membrane. The brain-substance generally was soft, putty-like in consistence, wanting in firmness. The left crus cerebri was almost in a fluid state of softening. The surface of the pons varolii, and also of the medulla oblongata, was softened. The basilar artery was thickened, and contained a thrombus. There was a large quantity of fluid in the ventricles.

Spinal Cord; anterior aspect.—On ripping open the dura-mater from below upwards no change is noticeable until the upper dorsal region is reached, when this membrane is found glued together with the soft membranes, and the whole mass firmly adherent to the cord. With the finger one feels a hardness about the size of a bean at the left side of the cord at the lower point of the cervical enlargement. Here it is found that for about 12 millimetres there is complete adhesion together of both hard and soft membranes: thence upwards for nearly 4 centimetres the pia mater is much thickened and adherent to the cord. At this part the cord, on section, is found to be of the consistence of rotten cheese. The whole extent of this softening is about two inches longitudinally. Above this again, and for the remaining 12 millimetres or so of the separated cord, the substance feels firmer, if not of natural solidity.

Posterior aspect.—The membranes appear natural, until we come to about 16 centimetres above the pointed extremity of the cord, where the dura mater is found thickened and adherent to the soft membranes. Slitting these up, they can with some little force be separated from the surface of the cord, and then this latter, as well as the exposed surface of soft membrane, wears an aspect of erosion, and is pink in colour. It is evident that one is tearing through recent inflammatory adhesion. This adhesion is very close for about 4 centimetres, and thence upwards, continues, though not so firmly, till we come to the cervical enlargement, where there is (as on the anterior aspect) a matting together of all the membranes. On cutting through the part where the hardness previously described is most pronounced, we find that it is of almost cartilaginous quality in section, and measures ·5 cm. in thickness. The cord is nearly fluid at this point, and scarcely a trace of the pattern of grey matter is to be seen on section.

There was no disease of the vertebræ.

My friend Dr. H. R. O. Sankey, of the County Asylum, Prestwich, was kind enough to examine the cord for me, and his report is as follows:—

"Transverse sections of the piece of cord which was sent to me were very difficult to cut, owing to the friability of the cord substance and the toughness of the thickened pia mater.

"*Microscopical appearance of Pia Mater.*—At the posterior part of the cord this was thicker than at the anterior, and where it was thickest it measured about $\frac{1}{10}$ inch. It consisted of connective tissue, but was not at all uniform in structure. At some places it was highly cellular; at others chiefly fibrous. The arrangement was very irregular, but at one part an appearance was seen in which the fibrous bundles were very regular in arrangement and parallel to the cord. But few vessels exist in this structure; those observed, however, were well developed, especially as to the adventitia, which passed insensibly into the general fibrous matrix in which they lay. In one case a very thin-walled lacuna-like space was seen filled with blood, which was uniformly coagulated, and had not long been in this position.

"The band of connective tissue which passes into the anterior fissure is greatly thickened, and contains vessels which are altered in the same manner as those of the substance of the cord (vide infra). The adhesion between the pia mater and the cord substance was broken down in the cutting of the sections, and cannot be made out, but it is probable that the entering trabeculæ are not thickened, but rather softened, like the cord substance.

"*White Matter of the Cord.*—This is throughout greatly altered. The changes are as follows:—

"1. Ascending fibres are in many parts only represented by homogeneous globules, staining slightly in carmine and logwood; while in others a few more or less normal fibres are seen; and in others only the shrivelled sheath of Schwann can be seen, devoid of axis cylinder, and filled with a homogeneous drop of myelin.

"2. Connective tissue is greatly increased. As to its cellular constituents, immense quantities of new cells, darkly stained, cover the field of view at every part.

"3. A granular débris fills the spaces left by these two elements. (N.B. Some of these appearances may be due to post-mortem decomposition, i.e. the state of the more healthy nerve tubercles and the granular appearance just described; the cells cannot be.)

"Vessels are large, tortuous, and the walls are covered by new cells, so that their structure is in places quite obscured. In some places, where it can be seen, it is healthy, and the vessels are all void of blood, or nearly so, and patent.

"*Grey Matter.*—Of the two cornua (anterior) the left is the more diseased. In this one, two or three nerve cells can be seen. These are circular bodies, shorn of all processes, showing a granular nucleus evidently in the last stage of degeneration. In the right cornua a larger number of cells are seen. They are more refractive than usual. They are in many instances devoid of processes, but retain them in other cases for a short distance. The posterior cornua are involved in the disorganisation. It is, however, impossible to say that they have suffered more than other parts, or that the right has suffered more than the left.

"All that can be made of the rest of the grey matter is that it contains too many nuclei, and is granular and opaque, and contains vessels surrounded by cells to a great extent. One vessel near the central canal contains an ante-mortem clot.

"*Summary.*—1. Fibrous thickening of pia mater.

"2. Infiltration of cord by new cells from vessels, and increase of existing connective tissue by migration or otherwise.

"3. Destruction of normal constituents.

"4. Post-mortem decomposition."

I take this to have been a case of inflammation of the internal layer of the spinal dura mater, of the arachnoid, and pia mater, the hard and soft membranes being agglutinated at the point described into a mass which was strongly adherent to the cord. The external layer of dura mater appeared to be unaltered. Dr. Sankey speaks of the tough mass as thickened "pia mater," but I imagine it is difficult to say with which of the membranes the hypertrophy described is to be especially associated, for they are all involved.

Charcot,[1] writing of hypertrophic cervical pachymeningitis, remarks that it consists of an alteration of membranes affecting more especially the dura mater, and notes that the cervical enlargement of the cord is in some respects a favourite seat of it. The alteration in the dura mater is the primary fact, but later on the cord and the peripheral nerves are involved. He thinks that the cases formerly described by Laennec, Andral, and Hutin, under the name of "hypertrophy of the spinal cord," properly belonged to cervical pachymeningitis; the swelling, which was really due to the membranes, being attributed to the cord itself. As a matter of fact, the cord, far from being hypertrophied, is squeezed from before backwards. The pia mater is affected, but much less than the dura mater, which may attain a thickness, he remarks, of 6 or 7 mm. (In the case above described the thickness, when the cord was fresh, was 5 mm.) The dura mater is usually altered in its entire thickness, as is proved by the adhesions which unite it outside to the vertebral ligament, inside to the pia mater. In the present case it will have been observed that the external layer of dura mater was free from change.

With this remarkable thickening and agglutination of membranes the cord at the part most affected by meningitis was broken down and disorganised. The condition was one of meningo-myelitis, which I should think was probably syphilitic in origin, for the following reasons:—1. The patient had suffered some eight or nine years previously from a venereal ulcer. 2. The autopsy showed the basilar artery thickened, and thrombosed; the crus cerebri, pons varolii, and medulla oblongata softened. The patient's age was about 35—too early for the arterial change to be reasonably ascribed to senile degeneration. The thickening (endarteritis) was precisely of the character which we see so frequently in the cerebral arteries in the sequel of syphilis. 3. During life, as will have been observed, an extraordinary improvement took place in the patient very shortly after he began to receive specific treatment, especially as regards his lower extremities.

In view of the state of the cornea in each eye, and the history of fits in childhood, it seems quite probable that

[1] 'Leçons sur les Maladies du Système Nerveux.'

the patient may also have been the subject of congenital syphilis.

I was anxious to discover whether in this case a microscopical examination would disclose thickening of the walls of the vessels in the substance of the cord. It appears to be very likely that in cases of syphilis there is frequently such a thickening of the walls of the spinal arteries as experience has proved to be the case as regards the intracranial arteries. Such a thickening was found in the basilar artery in the present instance, and it appeared, therefore, at the least probable that a similar condition might have existed in the vertebral arteries or their branches. I regret that it did not occur to me at the autopsy (which was made under circumstances of some difficulty) to remove specimens of the vertebral arteries for examination. The hardening of the cord was not accomplished very satisfactorily, and Dr. Sankey had some trouble in his examination. He found that the vessels, as well in the substance of the cord as in the connective tissue dipping into the anterior fissure, were large, tortuous, and their walls covered by new cells, but in only one instance was he able to observe the presence of an ante-mortem clot. This, however, isolated as it is, is of great importance. We are still unable to say anything certain regarding the stage at which this thrombosis of a thickened vessel occurred, or as to the relation in point of time between the inflammation of the membranes and the myelitis. Nor does the clinical history aid us in any attempt at deciding whether the cord or its membranes was first attacked with inflammation.

The paralysis of the arms in this man was throughout much more marked than that of the lower extremities. This was doubtless because in the former case it was due in great part to disorganisation of the centres within the cord, in the latter partly to the result of meningitis, partly also to the interruption of nervous impulses caused by the slow compression in the cervical region. The appearances would lead to the inference that under the specific treatment the recent inflammation of the membranes in the dorsal and lumbar regions had very much cleared up. The treatment came too late to affect to the same extent the more or less organised thickening of the membranes in the cervical region, although it

is surprising to note the amount of improvement which did take place in the state of the arms. I do not feel able to explain satisfactorily the reason of this.

The pains, as they were described by this patient, were characteristic of a membranous lesion, causing irritation, especially of the posterior roots of the brachial plexus. As Charcot has pointed out, these pains may be met with in intra-rachidian tumours, in Pott's disease, and in vertebral cancer, as well as in hypertrophic cervical pachymeningitis.

The contraction of the pupils which existed here is well known as an accompaniment of lesions of the upper part of the spinal cord.

According to Charcot, epileptic attacks sometimes manifest themselves periodically in cases of compression-lesion of the cord. Although this association is comparatively rare, he has been able to collect five cases of this kind. In the present case I believe that there was optic neuritis, although I am unable to speak so positively on this point as would have been desirable. The difficulty of using the ophthalmoscope was very great even when the pupil was dilated, as the patient presented ataxic movements, swaying to and fro on the chair, and jerking his arms occasionally, as he was seized with a dart of pain; and on account of the haziness of the media. I could find no trace of gumma in the membranes of the brain, but it is always possible that a formation of this kind may have been absorbed under treatment. In view of the probable existence of optic neuritis and the certain thrombosis of the basilar artery and the cerebral softening, I do not think we can reasonably refer the epileptic seizures in this case to the compression of the cord in the cervical region.

CASE II.—*Stiffness and aching of the right arm, followed, after three months, by loss of power in the left arm and leg; atrophy of interossei in the left hand; impaired cutaneous sensibility of right arm; tenderness on pressing 4th cervical spine; return of power in left limb, with more or less spastic rigidity and exaggerated reflex; family history of phthisis.*

A lady, æt. 26, married two years, never pregnant, was sent

to me in July 1879, on account of weakness and numbness in her limbs. She complained that her right arm was stiff and ached, the hand being numbed and tingling as though it had "gone to sleep." This had been the case since March last. Her attention had first been attracted by a change in her handwriting. There was nothing the matter with the right leg. About a week before I saw her, the left arm, which had been previously unaffected, had lost power, and continued feeble, without any such tingling or numbness as was experienced on the right side. At the same time, with this accession of weakness in the left arm, she found herself swerving to the left from weakness of the left leg. The left leg, like the arm, continued weak. If she held anything heavy in her left hand it trembled. The aspect of the limbs was unaltered, and there was no apparent wasting.

In the arms the reaction of the muscles to faradism was normal. In the lower extremities there appeared to be some diminution of excitability in the tibialis anticus group of each leg, but this was doubtful, as it was equal on the two sides. The electric current was felt much more strongly in the right than the left leg.

The patient complained of the lower part of her back aching in the morning on waking. She had never suffered from bad pain in the head, but at times had been troubled with a little neuralgia. She was thin and strumous-looking. On her father's side there is much consumption, six aunts and one uncle (paternal) having died of phthisis before thirty years of age. There is no history of injury. The catamenia are regular.

A month after her visit to me, on getting out of bed one morning she fell down, owing to a rather sudden increase of weakness in the left limbs. I saw her a day or two afterwards, when the left arm was very powerless, especially about the scapulo-humeral muscles. In walking she swerved always to the left. There was a difference in the sensibility of the two arms, a touch being felt more on the left than the right—the sensibility of the right being evidently dulled. If she attempted to hold a book in the left hand, it slipped through her fingers.

I found that deep pressure on the spine caused pain over the fourth cervical vertebra. In no other part of the spine was tenderness complained of when pressure was applied, and repeated examination always caused it at this point.

The recumbent posture and general attention to nutrition by food and drugs was the treatment adopted.

In October last her condition was noted as follows :—

The right arm is somewhat stiff, and there is want of perceptive power by the fingers and thumb. The grasp of this hand is appreciable, but weak.

In the left hand there is some slight power of grasp, though less than in the right, and no apparent affection of sensibility. In using the arm, as in attempting to hold a fork, there is trembling of the limb. There is some atrophy of the dorsal interossei in the left hand with over-extension of the first phalanges, and some atrophy also of the thenar eminence. The over-extension of the first phalanges causes the meta-carpo-phalangeal joints to project in the palm of the hand. The patient can touch the back of the head with the left hand, the movement being accompanied by tremor, and causing some aching in the arm. The right leg has continued to be unaffected. The left leg is still weak. In walking, this leg is stiff, and the foot is carried in a semicircle. She tends to walk on the inside of the foot. There is difficulty in dorsal flexion, the foot trembling when she attempts it. There is some increase of patellar tendon reflex in this leg. The general health is good.

I have to-day (March 5th, 1880) received an account of her present condition. The left hand, it is said, can be used perfectly well for all practical purposes, although it feels rather weak and inclined to tremble. The middle finger is still *en griffe*, but she thinks there has always been a tendency to that position. During the past winter she has suffered much from chilblains, and the fingers have not yet recovered their proper shape. The muscles of the arm ache if she extends it from the shoulder, and if she attempts to do her hair.

There is still a little lameness in walking, and the sole of the left foot has a tendency to scrape the ground.

At no time has there been any affection of the bladder or rectum.

In this case we may at least localise the lesion in the cervical region. The circumstance of the left leg as well as the arm being affected would imply that it is the substance of the cord (and not merely the membranes) in this region which is in some way involved. An affection of the anterior roots of spinal nerves alone might explain the powerlessness, and also the muscular atrophy in the left hand, but the affection of the left leg, and, still more, the rigidity in these limbs, pointing to secondary degeneration in the lateral column, appears to show that we must seek the cause of the paralysis and atrophy in lesion of the anterior cornua in the cervical region. The lesion is one which apparently affects especially the left side of the cord. There may be a tumour, or, what would be more probable, considering the family history, and the pain experienced when the fourth cervical spine is pressed, some vertebral caries with pachymeningitis, causing compression and some resulting myelitis.

In the case which follows, and which in certain respects presents several points of resemblance to the one just related, the conditions observed are consequent on an accident. The lad appears indeed to have "broken his neck."

CASE III.—*Injury to cervical spine whilst diving; complete paraplegia of all four limbs; recovery of power, with spastic condition of limbs from secondary degeneration.*

George H., æt. 18, by occupation a gardener, whilst bathing on July 18th, 1878, dived and struck his head against the bottom of the river in six feet of water. He was unconscious for a few minutes, and on coming to himself felt great pain in the back of the neck (level of third and fourth cervical spines), and found that there was total loss of power in all four limbs. Both hands were clenched after the accident, he says, and for a week he could not open them, nor could he move his head "any way." He says that he could feel "when they washed him."

About a week from the time of the accident he began to recover power gradually, in the right leg and arm first.

In January 1879 he could just manage to stand with a chair; in March he could walk a few yards by himself; and in April could dress himself. Since May he has been able to walk by himself with the aid of a stick.

When he applied at the National Hospital for the Paralysed and Epileptic as an out-patient, on July 9th, 1879, I noted that the electrical reaction of the left deltoid muscle was greater than that of the right to faradism, and less to the interrupted Voltaic current. The electrical reaction of the muscles of the legs appeared to be normal.

The patient was admitted into the hospital in October last, when the following note of his state was taken by Mr. A. E. Broster, Resident Medical Officer:—

"The spinal column shows nothing abnormal.

"The head can be moved perfectly in all directions. His breathing is natural, the diaphragm acting normally.

"*Upper limbs.*—There is no muscular wasting. The grasp of the right hand is 35 dynamometer, of the left 20. There is some rigidity about the wrists, especially the left, and the hands have a tendency to drop. During the movements of the arms there is some shaking. The muscular sense is normal.

"*Lower limbs.*—The legs present some 'clasp-knife' rigidity; the thighs resist abduction. His walk is stiff and slow, the left foot apparently dragging more than the right. Tickling the soles of the feet causes excessive reflex.

"The patellar tendon reflex is exaggerated in each knee. There is foot clonus in both extremities, but on the right side more than the left. He is able to walk about a quarter of a mile.

"The functions of the bladder are not impaired.

"There is no loss of cutaneous sensibility anywhere.

"The patient says that he has had no pains at any time in his arms or legs, except a little pain in the stiff shoulder-joints, and this only on movement."

In all probability in this case there was rupture of vertebral ligaments, and possibly also of intervertebral substance, with

hæmorrhage. There may have been displacement also or fracture of a vertebra, but the result leaves this uncertain. It is clear that in some way there was a sudden compression of the cord; and it is probable that, although the source of this compression has gradually been removed, the cord itself has sustained about the point indicated by the patient some permanent damage, which has been followed by secondary degeneration of the lateral columns.

CASE IV.—*Gradually increasing weakness of all four extremities; head rigidly fixed to one side; prominence of third cervical spine; slight paralysis of facial muscles and of palate; absence of movement of diaphragm; respiration, mainly upper thoracic; wide-spread loss of cutaneous sensibility; attack of double pneumonia; imminent peril to life; recovery from pulmonary affection; gradual increase in power of limbs, and recovery of movements of diaphragm.*

On May 14th, 1879, Mary Ann M——, æt. 13, was brought to the National Hospital, Queen Square, with gradually increasing weakness of the four extremities, and was admitted at once as in-patient.

The following notes were taken by Mr. A. E. Broster, Resident Medical Officer:—

"She came from a miserable hovel, where she was living in great poverty, and had been ill-fed. For a few months in the winter she had been in a situation as general servant, where she had to work very hard, and lift heavy weights. It was in December 1878, whilst thus employed, that she complained of sore throat, for which she applied a mustard-plaster, and then bathed her neck in cold water to remove the stinging which this occasioned. Next day she had a stiff neck. Two or three days later, rain came through the ceiling on to her head and body whilst she was asleep in bed. This was followed by pains in the neck and shoulders and the right knee-joint. About the end of January 1879 the right hand is described as having been swollen and blue. The thumb is said to have had little blisters on it. About a week later the right arm got bad, and then her shoulder became powerless. A few days more and the right leg began to fail, and at the end of March

she was forced to leave her place. A fortnight before her admission into the hospital the right arm had got, she says, to its worst, but the leg was still continuing to become more feeble.

"During the last week or ten days the left arm has failed, and the left leg has been getting numb and weak. For three weeks past she has been unable to walk by herself.

"The following was her state on admission:—

"The head is rotated to the right, and immovably fixed in this position, the long cranial axis being at an angle of 45°, with the inter-scapular axis on a plane parallel with and above the latter. The third cervical spine is thought to be somewhat unduly prominent, and slightly displaced to the left. Around and above it there is some hard thickening, apparently beneath the muscles. No tenderness is experienced except at a spot in a line with and below the right mastoid process.

"*Mental condition.*—Normal.

"*Special senses.*—*Smell* more acute on the left than the right side, tested by sumbul and assafœtida.

"*Sight.*—Reads 2½. Snellen left eye, 5½ right eye. Ophthalmoscope shows no change.

"*Hearing.*—Normal.

"*Taste.*—Somewhat deficient on both sides, but especially on the left.

"The upper lip has a somewhat expressionless look. When she smiles, the two angles of the mouth act evenly and fairly, but in voluntary effort, as e.g. in showing her teeth, there is decided weakness on both sides, most marked on the left. She cannot frown. She says she bites her right lower lip. Asked to shut her eyes, the lids fail to meet by ⅛-inch, but by a strong effort and the aid of other facial muscles she can approximate them. The reaction to faradism of the facial muscles is normal.

"The right masseter is thought to be somewhat stronger than the left, the horizontal movement of the lower jaw is good.

"The movements of the eyeballs are perfect. There has been no diplopia. Pupils are equal, moderate in size, and react well to light.

"She speaks clearly, there is slight tremor of the tongue.

Swallowing is imperfect, but liquids have never returned through the nose. The palate goes up more on the left than the right side. Uvula deviates to the left.

"*Tonsils* are large and somewhat reddened. A small ulcer seen on the left.

"*Laryngeal movements* normal. She is unable to move her head in any direction whatever.

"*Trunk.*—Respiratory movements. She says, 'I cannot take my breath so quick as I used to. Sometimes I cannot draw a long breath for two or three minutes, although I feel I want to.' She takes a number of short inspirations.

"When the hand is placed on the epigastric region, and the patient takes as deep an inspiration as possible, the abdominal wall is felt to fall in with the inspiration. There is very little movement of the ribs. There is no incontinence of urine or fæces.

"*Upper extremities.*—The right is very stiff, but no pain is caused by passive extension. There is 'clasp-knife' rigidity at the elbow-joint. She can just slightly flex this joint, and there is a faint power of grasp with the right hand.

"She can place her left hand on the top of her head, but the grasp of this hand is not sufficient to affect the dynamometer.

"The forearms, which are equal in size, measure 7½ inches at their greatest circumference. In the hands there is hyper-extension of the metacarpo-phalangeal joints, owing to weakness of the interossei, especially marked on the right side. There is no diminution of excitability to faradism in the muscles of either arm. From the drooping position of the wrists (especially the right) there is evidently weakness of the extensors.

"*Lower extremities.*—The patient can just stand; she cannot walk. There is marked 'clasp-knife' rigidity in the right much more than in the left. As she lies she can lift the left leg off the bed, not the right, and can draw up both legs, the left most strongly. Calves measure 10½ inches each. There is no foot clonus on either side. The patellar tendon reflex is exaggerated, and is more marked in the right than the left limb.

"*Cutaneous sensibility* is lost in the tips of the right fingers,

front of the right thigh, dorsum of the right foot, and the right shin. It is diminished over the trunk and extremities generally, from the neck downwards behind, and from 2 inches below the level of the clavicles in front, with the following exceptions. Along the ulnar border of the anterior aspect of the right forearm there is scarcely any diminution. Here there is distinct hyperalgesia to the induced current. The left upper arm is nearly normal as to its sensibility.

"There is no alteration of sensibility in the face, nor in the soles of the feet.

"The patient complains of no pain anywhere, but of a sensation of pins and needles in the forearms."

Family history.—Her mother has twice miscarried at the third month. The father has had no signs of syphilis, but had a gonorrhœa in youth. The other children show no signs of congenital syphilis. A brother died, four days before this girl attended the hospital, of rheumatism and bronchitis. Neither he nor any other member of the family suffered from a bad throat. There was no illness at the place where the patient lived as servant.

The patient had scarlet fever in infancy. Never had acute rheumatism, nor glandular swellings, nor injury by accident.

Viscera healthy. Temperature normal. The treatment was rest in bed and iodide of potassium. In the course of four days there was a good deal of improvement, both legs could be lifted off the bed, and there was some increase of power in the arms. The head, too, could be turned a little to the right and left, and bent forward a very little. The breathing remained as before, and Mr. Broster notes, "The pectorals and sterno-mastoids seem to do the work."

A gutta-percha support was fixed to the head and neck.

On June 12th it is noted that "during the last two days the patient has complained of pain in the right leg, shooting down from the groin to the knee, and there has been more difficulty in swallowing. There is wasting of the interossei."

Next day there was orthopnœa, the respirations very rapid, and entirely upper thoracic. There was some cyanosis. These symptoms increasing on the following day, the head-gear was removed, as it was thought it might interfere with

the action of the sterno-mastoids. Auscultation showed tubular breathing and crepitant râles along the base of the left lung, with harsh respiration in the right lung. There was very slight cough. The alæ nasi flapped. Her temperature was 101·4.

On the evening of the 15th her temperature was 103, the pulse 130, dicrotous. Respirations 36. She was very cyanotic. There were crepitant rhonchi throughout the left lung behind, and along the axillary border of the right. There was some delirium. Deglutition was much impaired, so that she could only swallow very small quantities of fluid. With great difficulty and gasping she stated that there was some pain in her head. At this time death appeared inevitable. After this there was gradual improvement in the respiratory system, but the loss of power in the extremities was at first more pronounced than it had been before the commencement of the pneumonic attack.

On the 18th I observed that the left side of the chest moved a little more than the right; the movement being mainly an elevation of the upper ribs; there was but very little lateral expansion. The epigastrium sank during inspiration.

On June 27th it is noted that the temperature is 100°; the respiration mainly upper thoracic, but with a little unilateral expansion. Over the base of the left lung the percussion note was dull, and there was increased vocal fremitus. Moist bubbling and crackling râles could be heard. On the right side there were a few small mucous râles along the posterior border.

Both legs could now be moved freely; each could be lifted off the bed and held extended for about thirty seconds.

By the middle of July all traces of the pneumonic attack had subsided. There was gradual improvement in the paralysed limbs. In the middle of September she had so far recovered that she was able to be sent to the Convalescent Establishment at Finchley, and the following note of her condition was then taken:—"She carries her head in the same 'side way' as on admission, and has gained no power as yet over its movements. She cannot look over her shoulder. The thickening previously described is very distinct around and over the third cervical spine. This spine can be felt distinctly prominent

beyond the other cervical spines. The weakness described in the facial muscles remains *in statu quo*, except that she can approximate her eyelids a little more than she could formerly. There is some deviation of the uvula to the left. The tonsils are large. The respiration is entirely thoracic, no movement of the diaphragm being perceptible.

"*Upper limbs.—Right.*—She can flex her elbow; the limb is slightly stiff. There is a perceptible, but not measurable, grasp of the hand. No distinct wasting of muscles. She can raise the arm to the level of the shoulder.

"*Left.*—She can hold up the arm fairly. The grasp of hand is not measurable. She can feed herself, and cut up meat, but cannot dress herself.

"*Lower limbs.*—She can stand and walk; the calves each measure ten inches in circumference. The walking is performed in a shaky manner, the trunk held stiffly, and the knees bent. She does not drag her feet. In rising from a chair she appears to have much difficulty, and requires a little impetus before she can get on to her legs.

"The patellar tendon reflex is exaggerated on each side. Foot-clonus is present, though not to a very marked extent, in both limbs—in the right more than in the left.

"*Cutaneous sensibility.*—On the left hand and forearm (except in the tips of the fingers, which are still numb) sensation is normal. On the left leg it is deficient.

"*Right upper limb.*—Sensibility is still deficient, though less so than formerly. On the right leg it is as good as on the face, where it is apparently unimpaired.

"For her own part, the patient says that the sensation in the left lower limb is better than it was; and that in the right leg, which used to be the worst, she feels better than in the left. The right leg, which was the weaker, is now, she says, the stronger. She says that a touch upon her back is felt quite right, but not so on the front of the body. She has no pains whatever, and no sensations of 'pins and needles.'

"Appetite good. She has control over the sphincters. Two months and a half later, whilst at Finchley, she was well able to get about by herself. The movement of the diaphragm had returned."

My first impression, on seeing this patient in the out-patient room, was that we had to do with one of those acute cases of nearly universal paralysis, the pathology of which remains obscure, and of which I have brought two marked examples before the Clinical Society of London. One had to consider the possibility also, in view of the history of "sore throat," that the affection might be diphtherial. Both these contingencies were disposed of by the observation of the fixed position of the head, and the discovery of the thickening about the third cervical spine. The case resolves itself, therefore, in all probability, into one of Pott's disease, high up in the cervical region. If this be so, the internal lesion (pachymeningitis), besides cutting off nervous impulses from the extremities, ribs, and diaphragm, apparently involves also to a certain extent the origins of the facial and glossopharyngeal nerves.

The pains were not strongly marked. Such as were present affected the head, doubtless through the occipital nerves.

The most interesting point in this remarkable case is the long-continued paralysis of the diaphragm. This paralysis was doubtless only partial, like the paralysis of the extremities and of the intercostals; but it was pronounced enough to give rise to the abdominal depression during inspiration mentioned by Duchenne as characteristic of paralysis of this muscle. Duchenne thus describes the condition. "At the moment of inspiration the epigastrium and the hypochondria are depressed, whilst, on the other hand, the chest dilates; the movements of these parts are reversed during expiration." Recovery from such an attack of pulmonary inflammation as this girl suffered must, in the circumstances of her case, be an exceedingly rare event.

DISTRIBUTION OF THE ARTERIES OF THE SPINAL CORD.

BY JAMES ROSS, M.D.,

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A COMPARISON of fortunate sections of the spinal cord has enabled me to map out pretty accurately the distribution of its vessels. The *anterior median*, the *posterior spinal*, and the *central arteries* are already well described in ordinary anatomical works, and need only be mentioned here.

The *anterior median artery* gives a series of small branches, which pass backwards in the anterior median fissure, to reach the anterior commissure; hence these vessels may be called the arteries of the anterior median fissure (Fig. 1, *a f*). Each of these vessels on reaching the anterior commissure divides into two main trunks, each of which enters the grey substance of the anterior horns, and which may be called the arteries of the anterior commissure (Fig. 1, *a c*). Each trunk subdivides into three branches, which, from their position, may respectively be named the anterior (Fig. 1, 1), median (Fig. 1, 1′), and posterior (Fig. 1, 1″) branches of the artery of the anterior commissure. The anterior branch curves forwards, and is distributed to the anterior and internal portion of the grey substance; the median is distributed to the lateral portion of the anterior horn, while the posterior is directed backwards to the posterior horn. The central artery also gives off an anterior (Fig. 1, 2) median (Fig. 1, 2′) and posterior (Fig. 1, 2″) branch, which are distributed respectively to the anterior, lateral and posterior portions of the grey substance. The median branches of the two main vessels, besides supplying the grey substance, are also distributed to

the pyramidal tract of the lateral column. The posterior spinal artery (Fig. 1, *p a*) gives off branches, which pass by the

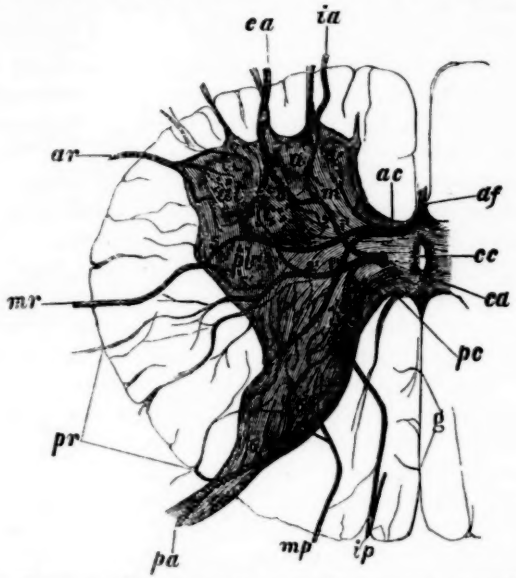


FIG. 1.—SCHEMATIC REPRESENTATION OF THE DISTRIBUTION OF THE BLOOD-VESSELS IN THE CORD.*

a Anterior median artery.
af Arteries of the anterior median fissure.
ac Artery of the anterior commissure.
1 Anterior branch.
1′ Median branch.
1″ Posterior branch.
ca Central artery.
2 Anterior branch.
2′ Median branch.
2″ Posterior branch.
pa Posterior root arteries.
6 6′ 6″ Arteries of posterior horns.
ia Internal anterior root artery.
ea External anterior root artery.
3 3′ Internal and external branch.
ar Antero-lateral branches.
4 Anterior branch.
mr Median lateral artery.
5 5′ Anterior and posterior branches.
pr Posterior lateral arteries.
ip Internal posterior artery.
mp External posterior artery.
g Arteries of the column of Gull.
pc Artery of the posterior commissure.
vc Vascular column of Clarke.
i Internal group of cells.
a Anterior group.
al Antero-lateral group.
pl Postero-lateral group.
c Central group.
m Median group.

side of the posterior roots to enter the grey substance of the posterior horns, where they subdivide into a variable number of small branches (Fig. 1, 6 6′ 6″), which may be called arteries

* I have to thank Dr. Young, of the Owens College, for the above diagram.

of the posterior horns. In addition to the vessels just described, a large number pass from the pia mater into the substance of the cord, and some of these are so large and so constant as to deserve special mention. Two of these run by the side of the bundles of fibres which constitute the anterior roots of the nerves, hence they may be called the anterior-root arteries. The branch nearest the median fissure may be called the internal anterior root (Fig. 1, *i a*), and the other the external anterior-root (Fig. 1, *ea*) artery. The internal artery on entering the grey substance joins the anterior branches of the first subdivision of the artery of the anterior median fissure and of the central artery. The external anterior-root artery on entering the grey substance subdivides into two branches, the inner (Fig. 1, 3) of which is distributed along with the vessels just mentioned, while the outer branch (Fig. 1, 3′) passes between what we may call the antero-lateral (Fig. 1, *a l*) and central groups (Fig. 1, *c*) of cells. A very constant vessel passes to the grey substance from the pia mater, at the point of junction of the anterior and lateral columns of the cord, and it may therefore be called the antero-lateral artery (Fig. 1, *a r*). On reaching the grey substance it frequently divides into three branches, one of which passes in front (Fig. 1, 4), another behind, and another into the substance of the antero-lateral group of cells. Another constant vessel (Fig. 1, *m r*) passes from the lateral aspect of the cord, and on reaching the grey substance it subdivides into two branches, the one of which passes in front and the other behind the postero-lateral group of cells (Fig. 1, *p l*), and this vessel may from its position be called the median-lateral artery. Small branches (Fig. 1, *p r*) pass at short intervals from one another through the posterior part of the lateral column, and, together with the median branches of the first subdivision of the artery of the anterior median fissure, and of the central arteries, supply the posterior part of the lateral columns; hence these vessels may be called posterior-lateral arteries. Two vessels pass from the pia mater into the substance of the posterior column, the one nearest the posterior median fissure, and which may therefore be called the internal posterior artery (Fig. 1, *i p*), passes between the column

of Goll and the posterior root zone; and after passing through about two-thirds of the depth of the posterior column, it curves outwards to reach the posterior grey horn. The other vessel may be named the external or median posterior artery (Fig. 1, *m p*); it passes into the substance of the posterior column at the middle of the posterior root zone, and on reaching about one-third the depth of the posterior column, it also curves outwards to reach the posterior grey horn, where it terminates. Small vessels (Fig. 1, *g*) pass from the pia mater, in the posterior-median fissure, into the substance of the column of Goll.

Another vessel, which may be called the artery of the posterior commissure (Fig. 1, *p c*), passes from the pia mater along the posterior margin of the posterior commissure, and winds backwards along the internal edge of the posterior horn.

With regard to the groups of cells in the anterior horns, I have, on the whole, followed the classification of Erb. These groups are the vesicular column of Clarke (*v c*), the postero-lateral (*p l*), and the antero-lateral groups (*a l*). The group which Erb calls anterior, I propose to call internal (*i*), because a very constant group of caudate cells is always observed near the anterior roots, which from its position is best distinguished as the anterior group (*a*). Also the group which Erb calls the median, I propose to call the central (*c*), because I wish to distinguish a very important area (*m*), which has hitherto not been described, and which from its position and connections is best denominated the median area. The cells of the median area are caudate, but they are much smaller than those of the other groups. This area is only present in the cervical and lumbar enlargements, the portions of the cord which regulate the movements of the limbs; its relatively larger size differentiates the anterior grey horn of the cervical enlargement of the adult human cord from the lumbar enlargement; in the human embryo at the fifth month and in the cord of the ox and dog, this area is of the same relative size in both the cervical and lumbar enlargements, and the anterior horns are under such circumstances almost indistinguishable; hence it may be presumed that in the median area are mainly organised the complicated movements which distinguish the hand of man from the anterior extremity

of the lower animals, so far as these movements are represented in the spinal cord. Other interesting facts tending to the same conclusion are that the cells of the median area in the human cord only assume processes after birth; while those of the other groups possess distinct processes at the fifth month of embryonic life.

The median area is entirely absent in the upper cervical and dorsal portions of the cord, and consequently the internal, anterior, antero-lateral, and central groups approximate, so that they are not always readily distinguishable. In these regions, however, an area is interposed in the human cord between the antero-lateral and postero-lateral groups, which possess some of the characteristics of the median area in the cervical region. The cells of this area are relatively small, they are comparatively late in development, and do not possess distinct processes at birth; and the area is entirely unrepresented in the cord of the ox and dog; hence it may be inferred that this area represents the additional organisation rendered necessary by the maintenance of the erect posture in man.

It may be readily imagined that in all inflammatory diseases the tissues in the vicinity of the vessels will be more liable than the remote portions to be inundated by effusion; hence the lines of the distribution of the vessels may be said to form lines of least resistance to disease. It is also worthy of remark that the development of the cells proceeds from the centres of the groups to their margins, and that in progressive degenerative diseases, the degeneration proceeds from the margins of the groups to their centres. To this law the median area in the lumbar and more especially in the cervical enlargement and that which lies between the antero-lateral and postero-lateral groups in the dorsal and upper cervical regions, are apparent exceptions. These areas, although containing cells, can hardly be called groups; they are probably areas where the marginal cell of the real groups meet. But whatever may be their nature they are the most vulnerable portions of the cord, and as they are the last to be developed, so they manifest the greatest liability to disease.

Critical Digests and Notices of Books.

Nothnagel on the Regional Diagnosis of Diseases of the Brain. (Topische Diagnostik der Gehirnkrankheiten. Eine Klinische Studie. Von Dr. Hermann Nothnagel. Berlin, 1879. Hirschwald, 8vo. pp. 626.)

This is an exceedingly able and important work, and worthy of the author's reputation as a physiologist and physician. It is an elaborate collection and digest of the more important facts and observations relating to the regional diagnosis of diseases of the encephalic centres, from the medulla oblongata up to the cerebral cortex and its individual parts. After recording and analysing the clinical facts bearing on disease of each particular region, the author sums up the conclusions which he considers they justify in a series of lucid diagnostic propositions.

Though naturally the work is largely of the nature of a compilation, yet the author displays much originality and critical acumen, and the whole bears impress of a mind thoroughly capable of dealing with every detail. It professes to deal only with the clinical aspect of the question. Physiological and anatomical considerations are reserved for treatment in a separate work. This will no doubt be looked for with interest by all who believe that anatomy, experimental physiology, and classical medicine ought mutually to support and shed light on each other. As to the propriety or even possibility of entirely abstracting from anatomical and physiological researches in a work devoted to the regional diagnosis of cerebral lesions, and therefore by implication determining the functions of the parts involved, there may be some difference of opinion. It may be possible, and perhaps advisable, to avoid entering

at length into these topics, but to exclude them entirely is not capable of being carried out; and in truth it may be asserted that but for the light of physiological research, clinical facts relating to cerebral disease would still be largely in a state of chaos. And certainly if the clinical evidence here furnished were all the basis on which the diagnostic propositions are based, we should have to regard the foundation as in many respects of an exceedingly slender character. Throughout the whole of this work, in fact, the experimental physiologist is clearly visible, and not unfrequently we find a tendency to draw conclusions, evidently in favour of some theory otherwise arrived at, from clinical facts which are neither sufficiently numerous nor of such a nature as to justify any conclusion whatever. That which renders analysis and conclusions from clinical facts alone extremely difficult and even hazardous, is the extreme complexity of the conditions, and the impossibility of distinguishing in all cases between direct and indirect effects of lesions. Though Nothnagel adopts Charcot's rules as to the necessity of excluding all cases where the lesions are of such a nature as obviously to produce general perturbation and far-reaching disturbance, and to consider as bearing on regional localisation only such lesions as are not likely to cause such indirect effects; yet this from a purely clinical point of view is very difficult to carry out. Brown-Séquard's reading of clinical facts has led, as is well known, to conclusions very different from those usually accepted and here advocated. And we see in our author but too frequently an assumption of indirect action where the clinical facts are at variance with his own hypothesis, and ignoring it when it might be highly important to take it into consideration. That the conclusions of Nothnagel will be more readily accepted than those of Brown-Séquard will depend not merely on the clinical evidence here adduced, but on evidence otherwise obtained, and under the circumstances indispensable.

Our author, however, occasionally forgets another very important principle, though he does acknowledge its validity, viz. to found no conclusions as to the effects of certain lesions unless these effects are constant or their absence satisfactorily accounted for. Negative instances, cases of lesions without

such symptoms, are sufficient to overturn a whole host of positive instances, where the facts seem to establish a causal relation between the lesion and the symptoms, while in reality the symptoms are dependent on some other condition. The history of the localisation of cerebral disease shows how precarious conclusions from clinical evidence alone have been, the conditions of strictly scientific evidence not having been fulfilled.

The manner in which our author in his first chapter treats of diseases of the *Cerebellum* strongly suggests the idea that his chief object is to prove by clinical facts that it is only the middle lobe that is concerned in the co-ordination of equilibrium, &c., and that the disorders characteristic of cerebellar disease only occur where the middle lobe is implicated.

The characteristic symptoms of cerebellar disease are a reeling and staggering gait and vertigo. These, however, are not absolutely pathognomonic of cerebellar disease, as they may occur from other affections of the central nervous system. The diagnosis must take other negative and positive phenomena into consideration. As accessory symptoms, but not due to the cerebellar lesion as such, we have the general phenomena indicative merely of intracranial disease, particularly tumours, viz. headache, vomiting, optic neuritis; and others produced indirectly, such as affection of speech, affections of cranial nerves, and general motor disturbances paralytic or spastic.

The special symptoms are in all cases referred to implication of the middle lobe, either directly or indirectly.

What the standard is by which the author discriminates between direct and indirect action is very difficult to discover from a study of the facts here presented, as well as from others which might be referred to. It has the appearance of being extremely arbitrary and dogmatic. It may be admitted, and clinical facts certainly favour the view, that lesions of the middle lobe are more frequently associated with cerebellar symptoms than when the lesion is in the lateral lobe, but this is very far from carrying with it the conclusion here maintained, that the middle lobe alone exercises those functions generally looked upon as cerebellar. Another explanation and one more in accordance with physiological facts, which,

though not mentioned, cannot be ignored, is that a lesion of the middle lobe is more likely to derange the cerebellum as a whole by affecting all its lobes at once. Our author even thinks that the facts—more particularly those relating to atrophy of the cerebellum—warrant him in advancing the hypothesis that the lateral lobes are "somehow related to the psychical functions." This hypothesis is, it is true, stated with some reserve; but, considering the kind of evidence offered, it would have been better perhaps to reserve it altogether.

Though several of the accessory phenomena, obviously dependent on indirect action on other structures, such as motor paralysis, spasms, &c., are mentioned as occurring in connection with cerebellar lesions, it seems somewhat strange that no particular notice is taken of the rigidity and tetanic-like seizures described so carefully by Hughlings-Jackson in connection with cerebellar tumours, so characteristic as to be almost pathognomonic. Even though indirect, these phenomena are of exceeding value in a diagnostic point of view, and too important to be dismissed among minor accessory symptoms.

The chapter on the cerebellum is certainly not the most satisfactory in the book.

As regards lesions of the *crura cerebelli*, the clinical facts do not allow us to decide positively as to the effects of lesions of the anterior or posterior peduncles respectively. Conclusions as to the special symptoms of lesions of the middle peduncles are also doubtful, owing to the fact that lesions are rarely accurately circumscribed. The most marked phenomena met with are distortion of the axes of the trunk, head, and eyes—particularly the latter—with vertigo and tendency to fall to one side. These our author looks upon as purely irritative phenomena, as in one case at least (Nonat's) the symptoms were not permanent, though the lesions remained. The rotation may be towards, or away from, the seat of lesion. This is not explained, though a fact of this kind might suggest doubt as to whether the symptoms can in all cases be regarded as purely irritative. Destructive and irritative lesions would be the converse of each other, and this may be the real ground of the

difference as to the side towards which rotation occurs. This, however, the author does not take into consideration.

Affections of the *Pons Varolii* are treated separately from those in which the medulla oblongata is also involved, though the two centres are very commonly implicated together.

Hæmorrhages in the pons are rare as compared with those in the hemispheres. Can we diagnose with certainty a hæmorrhage into the pons? Not always, says our author. The chief indication is the fact of alternate paralysis. Thus an apoplectic attack, with alternate paralysis of the limbs and face, with anarthria and dysphagia, points to the pons as the seat of lesion. Of great importance practically are signs of motor irritation, in the form of general epileptiform convulsions, considered pathognomonic by Ollivier. These convulsions Nothnagel explains by irritation of his so-called "convulsion centre," though why such a term should be employed, and an animal credited with a centre for the display only of pathological effects, it is not easy to comprehend without an abuse of physiological terms. That convulsions should readily occur from irritation of the pons, where all the sensory and motor strands of the encephalon are in such close relation, is a fact not difficult to understand without the questionable aid of such an objectionable term. These convulsions, however, are not constant, and Nothnagel does not agree with Ollivier in regarding them as of themselves pathognomonic. Vomiting is common, but not invariable. The state of the pupils in pontine hæmorrhage is one to which great importance has been attached. Very often they are contracted. Hence this condition, combined with complete muscular relaxation, simulates narcotic poisoning. The pupils, however, have been found dilated, or of normal size, and the reaction to light may also vary. Sudden death—fulminating apoplexy—is not uncommon from hæmorrhage into the pons, probably owing to indirect action on the medulla oblongata.

Neither the occurrence of albuminuria, nor of diabetes mellitus, though sometimes met with, can be taken as constant in pontine lesions. Similarly, also, as regards affections of circulation and respiration. These latter are to be ascribed to action on the medulla oblongata.

Prevost's idea that in pontine hæmorrhage the conjugate deviation of the head and eyes is away from the side of lesion instead of towards it, as in cerebral hæmorrhage, is not substantiated.

As a general result it is stated that a hæmorrhage into the pons can only be diagnosed with certainty "when there is alternate paralysis of the face and extremities. It may be considered probable when the apoplectic attack is accompanied by irritative motor-phenomena in the form of general convulsions, with contracted pupils, and when death occurs within a few hours. Under all other circumstances only conjectures are possible" (p. 105).

Embolism of the basilar artery is stated by Nothnagel not to occur. This, however, has been controverted by Gowers in a recent communication to this Journal. ('BRAIN,' No. VIII. "Illustrations of Diseases of the Pons Varolii.") Thrombosis is not uncommon. This may be rapidly fatal or lead to chronic softening, with "herd-symptome." These symptoms, which occur with hæmorrhagic or necrotic softenings, are very numerous.

Motor paralysis of the limbs on the side opposite the lesion may be seen—more marked in the lower than upper extremity—without affection of cranial nerves; or the cranial nerves may alone be paralysed; but more commonly there is alternate paralysis of the extremities on the opposite, and of cranial nerves on the same side as the lesion. But the face and limbs may be paralysed on the same side. This occurs when the lesion is nearer the crus cerebri; the alternate form occurring when the lesion is nearer the medulla. The facial paralysis in pontine lesions resembles peripheral facial paralysis, both as regards the diminution of faradic excitability, and as regards the reaction of degeneration to galvanism.

Besides the facial we may have affection of the trigeminus, abducens, (auditory?) and hypoglossal. The hypoglossal is affected next in frequency to the facial, and then the abducens. The affection may be on the side of lesion or on the opposite. Whether a conjugate ocular paralysis of the rectus externus on the same side, and rectus internus of the opposite side is characteristic of pontine lesion, is uncertain.

Double motor paralysis of the limbs is not common. Contractures in paralysed limbs often occur with stationary lesions, but not epileptiform convulsions. Occasionally ataxic symptoms have been observed.

Affections of sensibility also occur, either in the form of anæsthesia or hyperæsthesia in the paralysed parts. There may be facial anæsthesia on one side and anæsthesia of the limbs on the opposite.

The symptoms produced by tumours are very variable and difficult to distinguish from affections of the *basis cranii*. Some tumours remain latent; others cause alternate paralyses, or affections of cranial nerves, or disturbances of equilibrium.

The various facts here indicated are generalised in the diagnostic propositions at the end of the chapter, which is one deserving the highest praise, and which with its elaborate *résumé* and digest of cases will be of signal use to all neurologists.

The diagnosis of affections of the *crura cerebri* rests on the occurrence of paralysis of the oculo-motor on the side of lesion, and of the extremities on the opposite—the best marked instance of which is Weber's well-known and much-quoted case. Such a form of alternate paralysis can only occur with a lesion at the base of the brain. The situation in the crus is to be determined mainly by the mode of onset. If it occur suddenly, the crus is indicated—not otherwise.

Occasionally from tumours both third nerves may be implicated.

Lesions confined to the *corpora quadrigemina* in the form of hæmorrhage and softening do not exist. Hence conclusions as to the special effects of lesion of the corpora quadrigemina have to be founded almost entirely on tumours, affecting these ganglia in whole or in part, and for this reason very uncertain. All the published cases are considered in detail, and the following conclusions are based on them. Lesions of the anterior tubercles are almost always, if not always, associated with blindness. We may perhaps diagnose a lesion here, if we have along with the general indications of a cerebral affection sudden amaurosis with immobility of the pupils, without any abnormal ophthalmoscopic appearances. In cases of lesion of

the posterior pair we may have affection of the oculo-motor nerves. This, however, is dubious. The character of the affection is peculiar. Thus a bilateral affection of symmetrically functioning branches, such as double ptosis or double impairment of certain ocular movements, points to the corpora quadrigemina, especially if no alternate paralysis of the extremities exists. This bilateral affection may result from unilateral lesion. In connection also with lesions of the posterior tubercles, disorders of co-ordination, equilibration, &c., have been observed, similar to those of cerebellar lesion. This subject, however, requires further investigation.

Lesions of the *optic thalami* are treated at considerable length, as the obscurities and uncertainties surrounding the question require. According to the general plan followed, recent hæmorrhages and softenings, stationary lesions and tumours are successively discussed.

Hæmorrhages confined to the optic thalami, and not also implicating the corpora striata, are much less frequent than generally stated to be the case. There are no symptoms which enable us to diagnose with certainty a hæmorrhage into the optic thalami as such, at least at the beginning. The occurrence of tonic or clonic spastic phenomena, on which great reliance is placed by many observers, is not constant, nor are they pathognomonic of lesions of the optic thalami, as they occur also with hæmorrhages in the centrum ovale, cortex, pons, and crus cerebri. The symptoms met with in connection with chronic lesions are grouped under different heads, and illustrated by reported cases. (1) Some lesions, single or double, may be entirely latent; (2) in others then we have anæsthesia of the opposite side, without motor paralysis; (3) in a third group we have hemiplegia without affection of sensibility; (4) in a fourth we have hemichorea or athetosis, without loss of sensibility or motor paralysis; and in a fifth we have affections of vision. The symptoms, therefore, are exceedingly complex and contradictory, and the question is in how far the optic thalamus as such is related to the symptoms described.

It used to be taught by Saucerotte, and more recently advocated again by Lussana and Lemoigne, that the optic thalamus was more especially related to the upper extremity.

This, however, Nothnagel repudiates. He reiterates his view formerly expressed, that lesions accurately limited to the optic thalamus occasion no motor paralysis whatever.

Motor paralysis, if it does occur in connection with lesion of the optic thalamus, occurs most frequently when the lesion is in the middle third, and is explicable by implication of the internal capsule. Athetosis and post-hemiplegic chorea are, as is well known, ascribed by Charcot to lesion of the posterior part of the internal capsule, but Nothnagel is not satisfied that the optic thalamus is not also concerned in the causation. As to affections of cutaneous sensibility, he does not think they are related to lesion of the optic thalamus as such, but the question is not altogether definitively settled.

With respect to Crichton-Browne's view that lesions of the optic thalami are associated with abolition of reflex excitability, our author does not think it has been conclusively established, but he is disposed to accept it as probable.

The relation of the optic thalami to "psycho-reflex" movements is considered. In those cases where there is paralysis of volitional facial movements while the emotional continue, Nothnagel thinks the optic thalami and their cortical connections must be intact; while paralysis of the emotional with retention of the volitional in all probability will indicate lesion of these parts. This question, however, is one requiring further investigation. As to the relation of the optic thalamus, if any, to the muscular sense, we know nothing as yet at all definite.

The question as to the relation of the optic thalamus to vision depends mainly on two reported cases, Remy's and Hughlings-Jackson's. In the former it is doubtful whether there was crossed amblyopia or bilateral hemiopia; in the latter hemiopia was distinctly made out. Nothnagel, however, expresses considerable doubt as to whether the hemiopia can be regarded as dependent on the optic thalamus lesion, inasmuch as the same may result from lesion of the optic tract, and also from lesion of the occipital lobe—a question discussed below.

Vaso-motor disturbances are not specially related to optic thalamus lesions.

Our author would—but with reserve—in diagnosing a lesion of the optic thalamus lay special stress on affection of vision in connection with lesion of the posterior part, on the occurrence of hemichorea, athetosis or tremor, abolition of reflex excitability, and possibly of the muscular sense.

Lesions of the *corpora striata* are also discussed very fully, and on a consideration of the clinical facts he comes to the following conclusions.

Chronic lesions of the corpus striatum may be accompanied by motor, sensory and motor, and vaso-motor paralysis on the opposite side. Unless the lesion is very small the motor paralysis is a constant symptom. The hemiplegia may disappear if only the nucleus-caudatus or nucleus-lenticularis is affected. In cases of persistent paralysis—which occurs when the internal capsule is injured—secondary contracture frequently sets in. The motor paralysis usually affects both extremities, the lower facial region, and also to some extent the thoracic and trunk muscles. The hypoglossus is either not affected, or only temporarily, very seldom permanently. In rare cases only the extremities, or the facial movements alone are affected. It is impossible to distinguish between lesion of the nucleus caudatus and nucleus lenticularis. Motor paralysis is the only symptom where the lesion is confined to the anterior part of the corpus striatum, or the region supplied by the lenticulostriate arteries. In some cases hemianæsthesia accompanies the hemiplegia. This hemianæsthesia is characterised by the implication of the special senses along with cutaneous sensibility on the side opposite the lesion; but the hemianæsthesia may be confined to cutaneous sensibility alone. Usually hemianæsthesia and hemiplegia co-exist. Only exceptionally does the latter disappear, leaving the anæsthesia. The existence of hemianæsthesia points to lesion of the posterior part of the internal capsule. The lesion is also frequently accompanied by vaso-motor disturbances on the paralysed side.

Hemichorea often occurs along with anæsthesia, but its relation if any to the corpus striatum is doubtful.

Centrum Ovale.—In considering and localising lesions of the centrum ovale Nothnagel arrives at essentially the same conclusions as Pitres (Lésions du Centre Ovale). He, however,

suggests some modifications of his "sections" and nomenclature—not, however, of much importance.

Lesions are latent when they occupy the occipital, sphenoidal, and frontal (anterior and middle) sections. Tumours in these regions may also remain latent as regards special symptoms, only those characteristic of intracranial tumours in general existing.

Even if special symptoms exist in connection with lesions of the centrum ovale, it is not possible to fix the seat of lesion with certainty, as the same symptoms may occur from cortical lesions, or even from lesions of the corpus striatum. The special symptoms, which occur only with lesions in the frontoparietal region, are either general hemiplegia, or monoplegia, as in cortical lesions. Our author considers it doubtful if convulsive spasms occur when the lesion is strictly confined to the centrum ovale, and does not implicate the cortex; but early rigidity may occur, as argued by Pitres, from irritation of the cortico-striate medullary fibres. We have no sufficient material for forming any conclusions as to the relation of lesions of the centrum ovale to trophic or vaso-motor disturbances. Aphasia may occur in connection with lesions in the medullary fasciculi of the third left frontal convolution.

Cortex Cerebri.—In treating of lesions of the cortex our author again expressly states that he founds his conclusions on clinical facts alone—entirely abstracting from physiological considerations. His conclusions as regards affections of motility agree in all essential respects with those of Charcot and other writers on cerebral localisation. That is, that in lesions strictly cortical affecting the anterior and posterior central convolutions, and paracentral lobule, we have either hemiplegia, or monoplegia, according to the position and extent of the lesion in these regions. As regards the exact locality of the lesions causing the different forms of monoplegia, there is little or nothing requiring special mention in connection with the literature of this subject.

The cortical position of the lesion is to be diagnosed not so much from the paralysis itself but from the accessory phenomena, such as local or generalised irritative phenomena, &c.

Hemiplegia with aphasia points to cortical origin; also hemiplegia associated with ptosis.

Nothnagel, however, does not accept the view advocated by Landouzy that ptosis indicates lesion of the parietal lobule (angular gyrus). In reviewing the cases which have been brought forward as destructive lesions of the motor zones without paralysis, he holds that there is not a single unequivocal case on record. Either the position of the lesion is not accurately given, or the nature of the lesion is such as not necessarily to destroy or entirely abolish functional activity. He justly ridicules a collection of cases by Lussana and Lemoigne as of no scientific value whatever.

Before passing from Nothnagel's consideration of the motor affections in connection with cortical lesions, we may observe that paralysis of the tongue from cerebral lesions is spoken of as paralysis of the hypoglossus, and similarly in regard to other monoplegiæ. By the term paralysis of the hypoglossus a peripheral paralysis is apt to be suggested, though not intended. It would be well to avoid terms which are likely to create confusion and misconception.

The affections of sensibility which our author attempts to connect with lesions in certain parts of the cortex are affections of the muscular sense, and affections of sight—particularly hemiopia. The evidence adduced on these heads, however, is not of a very satisfactory character.

He admits that affections of cutaneous sensibility have no relation to cortical lesions, and yet on the strength of two or three cases, complicated either with cutaneous paræsthesia or anæsthesia, and not one of which can be regarded as admissible in evidence, the lesions being either multiple, or extending deeply into the brain-substance, he ventures to conclude that probably affections of muscular sense are due to lesion in the parietal region of the cortex, a region near, but not identical with, the motor regions—a region, likewise, in which lesions are frequently if not generally found to be latent.

In formulating such conclusions our author seems to violate the principles by which he professes to be guided in reading clinical facts.

The very cases here related may be interpreted much more in accordance with established clinical facts, by lesion direct or indirect of the posterior part of the internal capsule and its neighbourhood.

Our author refers to two distinct affections of sight in connection with cortical lesions, (1) double hemiopia, (2) crossed amblyopia.

He attempts to bring hemiopia in relation with lesion of the occipital lobe—the retina being affected on the side of the lesion. That lesions of the occipital lobes may be altogether latent is also admitted.

The cases on which the relation between hemiopia and lesion of the occipital lobe is founded are far from satisfactory. In Pooley's case there was also a lesion in the optic thalamus; in Hirschberg's there was a tumour reaching to the optic thalamus; Wernicke's was a case of multiple lesion, both cortical and medullary; in Baumgarten's there was also lesion of the optic thalamus; and Nothnagel's own case was one of diffuse and multiple lesion in both hemispheres.

On material such as this it is surely hazardous to found conclusions.

Moreover, when we consider that it has been clearly shown that hemiopia occurs from lesion of the optic tract, and from lesion of the optic thalamus posteriorly, we require very stringent evidence that these parts were free from implication, directly or indirectly, before we can admit any causal relation between occipital lesion and hemiopia as being made out. In a case recently brought before the Soc. Méd. of Geneva by J. L. Prevost, in which hemiopia was very definitely determined, there was extensive occipital lesion, but also lesion of the posterior part of the optic thalamus and external geniculate body.

Though Nothnagel seems to be very doubtful of his own conclusions in his analysis of the facts, he is not so careful elsewhere, and one might be led to consider the proposition established, which at present at least it is not. While he with some degree of confidence connects hemiopia with lesions of the occipital lobe, he does not offer any suggestion as to the locality of the lesion which produces crossed blindness. This

symptom has been chiefly observed by Fürstner in general paralytics.

Nothnagel thinks that the connection between "word-deafness" and lesion of the superior temporo-sphenoidal convolution fairly well-established. As regards aphasia, his conclusions are essentially those of Kussmaul. We have no definite clinical facts enabling us to connect affections of smell and taste with cortical lesions. Similarly as regards trophic and vaso-motor disturbances. Psychical disturbances are as yet beyond localisation.

As to the effects of lesions limited to the *cornu ammonis* we at present know nothing. The reported facts as to the occurrence of sclerosis and atrophy of the hippocampus in epileptics are briefly reviewed, but no causal relationship is considered to be made out. There are likewise no symptoms enabling us to diagnose lesion in the *claustrum* or *external capsule*.

As to the *ventricles*, there are no definite symptoms indicating effusion in one or other as distinct from effusions elsewhere. A diagnosis of lesion of the fourth ventricle may be made if after an injury to the head there should appear diabetes mellitus or insipidus, with general cerebral symptoms such as headache, vertigo, vomiting, and possibly also slowing of the pulse and mental hebetude. The diagnosis will of course be rendered more precise by the occurrence of symptoms special to lesions of the pons and medulla already described.

Affections of the *basis cranii* are very variable and complex. Apart from general indications of intracranial disease, the diagnosis must be founded chiefly on the implication—destructive or irritative—of the cranial nerves, unilaterally or bilaterally, according to the position of the lesion in the anterior, middle, or posterior fossa; together with indications of affection of the cerebro-spinal tracts.

Lesions confined to the anterior fossa implicate the olfactory, and also the optic if they extend backwards.

In the middle fossa the optic, third, fourth, and sixth cranial nerves are specially liable, and also the olfactory if the lesion extends forwards. The facial can only be affected if the lesion extends into the hiatus of the aqueduct of Fallopius.

Lesions in the posterior fossa may affect the fourth, sixth,

and seventh (auditory and facial), the eighth and ninth. The third may also be implicated if the lesion extends forwards towards the middle fossa. With lesions of the posterior fossa also disorders of co-ordination specially occur.

Lesions of the *pituitary gland* cannot be distinguished from other affections of the middle fossa. Nor can *aneurism of the basilar artery* be made out with certainty or distinguished from lesions of the pons from other causes.

At the end of the work there is an "Ueberblick über die Herdsymptome," which is a kind of *catalogue raisonné* of the various facts detailed in the body of the work. In this there is a tendency to speak somewhat more positively than the facts and considerations in the previous part of the work justify. But though exceptions may be taken to not a few of the propositions stated in this book, it is without doubt a splendid contribution to the literature of cerebral disease, and will take its place as a standard work of reference among all who have to deal with diseases of the nervous system.

DAVID FERRIER.

The Crayfish. An Introduction to the Study of Zoology. By T. H. HUXLEY, F.R.S. C. Kegan Paul, 1880. International Scientific Series XXVIII.

THE publication of this carefully prepared and admirably illustrated volume should be a subject of congratulation to a large number of different kinds of readers. It will supply a need which the higher class of so-called general readers have often felt;[1] an exposition of the current theories of morphology and zoology, so illustrated by a reference to concrete facts as to enable the reader to pass his own judgment on the character of the explanations offered; in some such category as this we find, too, the medical practitioner whose knowledge of comparative anatomy has grown somewhat hazy, although he still

[1] A class which may, in Mr. Mark Pattison's words, be said to have for their aim "to improve faculty, not to acquire knowledge" (see his just published 'Life of Milton,' p. 210).

retains an affection for the science which throws so much light over the organism with which it is his especial duty to deal. The serious student, commencing morphology and acquainted with the first elements of dissection, will find that for him its chief value lies in the way in which zoological facts and anatomical characters are used to illustrate the processes by which modern naturalists attempt to explain the phenomena of living creatures. The researches of Mr. Ward, to which it is purposed to call especial attention later on, will suggest that the commencing physiologist, unknown to fame, and prevented, by the ill-directed activity of the Legislature, from experimenting on the vertebrated animals, may find in this volume not only many suggestive hints as to the physiological processes of the crayfish, but, further, that groundwork of anatomical details which will equip him for the work of resolving some still pressing physiological problems by the aid of this quite sufficiently complex organism.

There is no need to say that the book is well written, the style concise, the expositions clear; but it is a satisfaction to add that there are no attempts at fine writing or any humorous interpolations, while the woodcuts are eloquent of the care that has been bestowed upon them.

Commencing with an account of the 'Natural History of the Common Crayfish,' the author insists, at the outset, on the fact that "science is common sense at its best; that is, rigidly accurate in observation, and merciless to fallacy in logic;" this is urged from several points of view; it is shown how a common knowledge of nature grows into physical science, and how its various stages of growth can be daily exemplified from our knowledge of those around us; it is used as an argument for technical terms, and we would recommend those, who look upon science as a mere matter of hard names, to read what comes before and what succeeds the following sentence:

"Existing signs may be combined in loose and cumbrous periphrases; or new signs, having a well-understood and definite signification, may be invented. The practice of sensible people shows the advantage of the latter course; and here, as elsewhere, science has simply followed and improved

upon common sense." Whatever be the common sense possessed by those to whom this quotation is addressed, they must be careful lest they are not outstripped by the subject of this volume; we learn that not only are crayfishes intolerant of great heat, but that they affect those parts of a river which run north or south, because they yield more shade from the noonday sun than those parts which run east and west. In the winter they form burrows in the banks, and these burrows are deeper when the waters are liable to freeze. It seems, however, that from the human point of view, at any rate, the crayfish carries his common sense a little too far, perhaps because he cannot convert it into science; "crayfishes are guilty of cannibalism in its worst forms; and a French observer pathetically remarks that the males 'méconnaissent les plus saints devoirs;' and, not content with mutilating or killing their spouses, after the fashion of animals of higher moral pretensions, they descend to the lowest depths of utilitarian turpitude, and finish by eating them."

To come to the nervous system itself. As is well known, all animals which are known as invertebrata are distinguished from those which are called vertebrata (here, of course, including, as a degenerated branch, the tunicata) by the obvious fact that their central nervous system is "ventral" and not "dorsal," or, in other words, that they are "neuropodous" and not "hæmapodous"; the "ventral ganglionic chain" is in animals which, like the crayfish, are made up of a number of successive segments arranged typically in the same metameric fashion as the appendages and some other of the organs of the body; it is, however, rare to find in the adult that the masses of ganglionic matter retain their primitive disposition; some of the lowlier crustacea would appear to do so, but in all the higher forms there is apparent, especially in the more anterior region, a more or less striking concrescence of a certain number of ganglionic masses; in a common crab (*Carcinus mænas*) this fusion is carried, for example, to an extreme, and the whole of the ganglia of the ventral cord are fused into a single mass. The crayfish has thirteen distinct ganglia to its twenty segments, and of these the first is the brain or supra-œsophageal mass; the second or sub-œsophageal ganglion is

apparently composed of five primitively distinct masses, for it sends off nerves for five segments, while the supra-œsophageal ganglia are, as in so very many of the forms allied to the crayfish (other arthropoda), formed by the fusion of three others.

These ganglia are connected together by a pair of commissural cords of varying length, while they give off a number of fibres either to the muscles, or to the organs of sense, or to the integument generally; and the only important difference between them seems to lie in the influence which the supra-œsophageal ganglia have over the rest. When these are extirpated we find that, as Professor Huxley states, the creature, which, when uninjured, always attempts and ordinarily succeeds in regaining its proper position if it be placed on its back, is now no longer able to do so; "its limbs are in incessant motion, but they are 'all abroad;' and if it turns over on one side, it does not seem able to steady itself, but rolls on to its back again." If, previously to the operation, anything is put between its own chelæ it may try to use it as a means to turn over; but after extirpation of the ganglia it seizes the object and tries to swallow it; if the object is pulled back, the chelæ of the other side are brought to its aid and a co-ordination of movements is strikingly exhibited; this co-ordination may be destroyed by an ablation of the thoracic ganglia. These observations are sufficient to show (1) the influence of the cerebral ganglia, and (2) the independence of the succeeding portion of the nerve-cord. The results attained to by Mr. Ward go very much further, and it will not be inappropriate here to draw attention to the results which, in his preliminary notice, that observer has put out.[1] The first inference to which his researches have led him is this, there is no decussation of the longitudinal fibres in the cray-fish; this dictum will be found to be supported by the fact that division of one of the supra-œsophageal commissures is followed by an enfeebled nervous activity on the same side; not only does it happen that feeble, if any, responses are given to "considerable

[1] 'Observations on the Physiology of the Nervous System of the Crayfish.' By James Ward, M.A., Proc. Royal Soc. March 6, 1879. (P.R.S. xxviii., p. 379: Communicated by Dr. M. Foster, F.R.S.) See, also, 'Journal of Physiology,' II., pp. 214-228.

excitation" of the antennæ and eye-stalks, but a conspicuous want of symmetry is observable in the action of the abdominal appendages, and "the chelæ during progression show a bias towards the sound side."

The second result is of considerable importance. It was found that the spontaneous, or "volitional" activity of the *animal as a whole* depended upon the functional presence of the supra-œsophageal ganglia. This was shown not only by the positive fact that the animal exhibits spontaneity and purpose so long as one commissure remains intact, but by the negative results observed when both are cut. These results must be quoted in Mr. Ward's own words:—

"When both commissures connecting the supra- with the sub-œsophageal ganglion are divided, everything of the kind [spontaneity and purpose] disappears, save that occasionally the antennæ are waved about in the normal fashion, though much more feebly. The animal lies on its back, the maxillipedes, the chelæ, and the first three pairs of legs, for the most part, swinging slowly to and fro in perfect *tempo;* not, however, as the swimmerets do, both sides synchronously, but with the movements of one side alternating with those of the other. . . . . The feeding movements are a perfect mimicry of the movements made when food is actually seized. These last appear to be in all respects perfectly co-ordinated; so much so, indeed, that the chelate legs will wait their turn to pass their morsel to the mouth when scraps are placed in all of them at once. But neither they, nor the chelæ, nor the posterior maxillipedes show any selective power, even the animal's own antennæ being seized. The first evidence of taste appears when the food gets within the gape of the mandibles." When, in such condition of its nervous centres, the animal attempts to walk it is found that after a few slow and tottering steps it rolls helplessly on to its back. The results just now detailed are also sufficient to show that the supra-œsophageal ganglion presides over the inhibition of the "aimless and wasteful mechanical activity of the lower centres," and over the power to maintain equilibrium.

When the supra-œsophageal ganglion has been put aside, the next question is as to the special influence which the sub-

œsophageal ganglion may possess. This, again, may be looked at from what has been called the positive and the negative aspect. As to the former, it is, as Mr. Ward points out, obvious that "the sub-œsophageal ganglion is the source of a considerable amount of motor energy," for not only is there, as already detailed, an amount of movement still possible when these centres are still connected with the posterior portions of the cord, but it was found on experiment that the posterior maxillipedes still exhibit sufficient energy to raise the creature several times, and preening movements of the hinder limbs are well exhibited; better, indeed, than before. On the other, or negative side, we find such evidence as this: "The chelæ sprawl helplessly on either side, and the legs are for the most part doubled up under the body." The chelæ, too, are not always successful in passing food to the mouth, and when they do get it there they still do not let it go. The sub-œsophageal ganglion would seem, therefore, to be the centre for the feeding movements.

Our quotations from Mr. Ward are even already not inconsiderable; but it is impossible to put into less concise language the differences between the crayfish and the frog as enunciated in his next paragraph:—

"There is much less solidarity, a much less perfect *consensus* among the nervous centres in the crayfish than in animals higher in the scale. The brainless frog, e.g., is motionless except when stimulated, and even then does nothing to suggest that its members have a life on their own account; whereas the limbs of a crayfish, deprived of its first two ganglia, are almost incessantly preening, and when feeding movements are started the chelate legs rob and play at cross purposes with each other as well as four distinct individuals could do."

Here we must leave Mr. Ward, but we cannot do so without expressing a sincere hope that a full account of his important researches will soon be published,[1] nor without returning again to the point at which we hinted at the commencement that other physiologists will concern themselves with a creature

[1] A notice by M. Yung in the 'Comptes Rendus,' lxxxviii. p. 347, affords an independent support to several of Mr. Ward's conclusions.

in which the phenomena of inhibition seem to be particularly easily accessible.

The general reader already referred to will probably be a little astonished at the heading of page 89. The ordinary human being has, for no reason at all, either positive or negative that we have ever been able to discover, except from the strength of his position as a lord of creation, assumed to himself the especial and peculiar possession of a mind; the term, therefore, "the crayfish mind" will, no doubt, excite his antipathies, and the fact that the question of the crayfish having or not having a mind is left an open question will hardly console him. The various steps of a reasoning process, and the stage at which this becomes logical are, nevertheless, most admirably stated in a few short paragraphs by Professor Huxley, and the whole question summed up by pointing out that we could only have a "positive assurance" that the crayfish possesses consciousness by being a crayfish ourselves. Seeing no expectation of this, the phenomena associated with the activity of nerve and muscle are carefully explained, and form the basis for the important physiological lesson that so-called spontaneous movements do not really cause themselves, and that what, at any rate, the crayfish "does at any moment would be as clearly intelligible, if we only knew all the internal and external conditions of the case, as the striking of a clock is to any one who understands clockwork."[1]

Some points which especially interest nerve-physiologists are, however, altogether passed over. This, no doubt, is completely in accordance with the aim which this experienced expositor of scientific facts had definitely set before himself; but it should be pointed out that, so far as analogy, at any rate, is concerned, it is a considerable advantage to the student of the vertebrata to know that there is a visceral system of nerves in the non-vertebrated animals. The ganglia on the dorsal surface of the stomach, and the two nerves given off from the œsophageal commissures, to say nothing of the plexus

[1] A sentence of Mr. Pattison's ('Milton,' p. 174) is well worthy of being quoted in this connection: "That in selecting a Scriptural subject he was not in fact exercising any choice, but was *determined by his circumstances, is only what must be said of all choosing.*"

which is reported to be connected with the last abdominal ganglion, are so far of importance that they remove a difficulty from the student, who so frequently learns nothing of a sympathetic system till he comes into contact with vertebrated animals, and they seem from their origin to have a still higher significance, if the view to which Mr. Balfour is evidently inclined shall be justified by further investigations; the view, namely, that intestinal branches are developed on the main nerve stems of the thoracic and abdominal portions of the central nervous system, and that the ganglia developed on these become connected by a longitudinal commissure.[1]

This, of course, would be by the way in any other journal than 'BRAIN.' Having said this, we may, however, draw attention to one or two interesting points in nerve-muscle physiology which have recently resulted from the observations of continental observers:—

(1) When the visceral nerves passing off from the commissural cords are directly excited, the movements of the heart are accelerated; but if the thoracic ganglia (which communicate through the supra-œsophageal ganglion) are excited, the heart's beat is retarded (Yung). These first results may, perhaps, be not unfairly compared with the influence of the spinal *accelerator* nerves in the vertebrata.

(2) The tail-muscles of the crayfish soon lose their power of contraction when they are rapidly affected by a number of electric shocks; this is in keeping with the fact that the animal never swims for long at a time. (Richet.)

(3) The muscles of the great chelæ may retain their contractility for as long as four days after separation from the body; this, too, is to be correlated with the creature's well-known habits. (Richet.)

(4) Motor excitations pass more slowly along a lobster's motor nerve than along that of a frog; the proportion, per second, being six to twenty-seven mètres. (Frédéricq).

These, and observations like these, which might assuredly be multiplied, are, from a general point of view, of deep significance as illustrating, just as much as every series of

[1] 'Elasmobranch Fishes,' p. 173.

investigations into morphology or physiology does, the deep truth of Goethe's words:—

"Alle Gestalten sind ähnlich, doch gleichet keine den andern."

And they lead us, as they ought to lead us, to examine with respectful attention that theory of Evolution which Professor Huxley has done so much to render intelligible as well as to demonstrate as reasonable.

Let us now see how he deals with what he finds in the crayfish to support the doctrine he has for many years, through good report and ill, so manfully defended. No division of the animal kingdom is, in this connection, more interesting than the crustacea; for from no group have more convincing argumentations in favour of the hypothesis of Evolution been drawn. It is now some sixteen years since one of the most philosophical and careful of modern naturalists, Fritz Müller, tested the views of Mr. Darwin by an especial reference to the characters of the crustacea. His essay, entitled 'Für Darwin,' has been translated into English, and is continually in the hands of those who are interested in the discussion. As compared with his work, which to the student is of inestimable value, the contribution now offered by Professor Huxley is especially interesting. Fritz Müller's work was written in the days now fortunately quite gone by, when the attacks on the great modern prophet of Evolution were of a character which, justly enough, roused the spirit of his disciples. In the discussion under that aspect Professor Huxley took his share; but in the present volume we learn even more convincingly than we do from those remnants of the past style of "anti-Darwinian" criticism which now and again appear in our monthly magazines that the battle is really over, and that, till some formidable rival of greater scientific pretensions shall appear, the doctrine of Evolution has gained the day. In earlier times Mr. Huxley has known how to use all the weapons of scientific controversy; to-day he has found it safe to lay aside all armour, and, like his master in this great matter, he has dared to expose even his weak points. The discussion, therefore, to which he invites his readers in the last chapter of the volume is not only interesting from the way in which difficulties are

pointed out, but it is valuable as affording evidence that in matters more or less hypothetical the naturalist now, as in the pre-theoretical days which some still profess to admire, has no other aim than that of coming, after a time of struggle, to some clearer apprehension of the real meaning of natural phenomena. It will be right here to quote our author:—

"The preceding discussion must rather be regarded as an illustration of the sort of argumentation by which a completely satisfactory theory of the etiology of the crayfish will some day be established, than as sufficing to construct such a theory. It must be admitted that it does not account for the whole of the positive facts which have been ascertained; and that it requires supplementing, in order to furnish even a plausible explanation of various negative facts." (p. 333.)

The positive fact here referred to is this. So far as we know their characters, the East-American genus (*Cambarus*), in which there is one gill less than in our crayfish, has among the crayfishes (*Astacus*) its most closely allied form in the crayfish of the Amurland and of Japan; while the West-American forms are more like the Ponto-Caspian form. No information that we have at present of the disposition of land and sea in earlier geological epochs can yet explain this difficulty. An explanation of the negative fact that crayfishes are not found in certain regions is afforded us by the idea that physical obstacles, of which we have no knowledge, prevented the primitively marine ancestor from ascending certain rivers, and by the not unlikely suggestion that in certain rivers the struggle for existence was more than the invader could sustain.

Our space does not permit us to follow the author into his account of the fossil forms allied to the crayfish; into the accurate and instructive definition which he gives of the meaning of those difficult abstractions, species and genus; or into the history of the development of the special form under description. It will be interesting, however, to draw attention to a single example, which will, perhaps, suffice to show that while embryologists are accused of weaving phylogenies without end, they are, in truth, beset with many difficulties, the partial resolution of which has at least to be accomplished before they can attain to any consistent theory of develop-

ment. As in many other forms, part of the large intestine of the crayfish and of the lobster is formed by an inpushing of the ectodermal lining of the body; in the lobster it is estimated that one-sixth, and in the crayfish fifteen-sixteenths, of the large intestine have their origin from the outer layer of the body. Differences such as these are continually met with by the student of development; they are discussed with his co-workers, and it is the grand results of continuous and comparative labours which the world is apt to denominate as the results of a superficial examination. The more deeply we study the animal world, the more do we see how apparently interminable these differences are. They are hard enough to cause at times a feeling of despondency, which is only relieved by a generous sympathy; this naturalists have often, though hardly often enough, extended to their fellows. Now, however, we shall be able to look to a wider circle of friends; for no intelligent man or woman will read 'The Crayfish' without having excited in them a deeper interest than they have yet felt in the details of animal organisation, and in the labours of its investigators. From this important point of view the work now in hand is especially to be welcomed.

F. Jeffrey Bell.

Pseudo-hypertrophic Muscular Paralysis. A Clinical Lecture. By W. R. Gowers, M.D., F.R.C.P. London: J. and A. Churchill, 1879.

This volume contains a reprint of a lecture which appeared in the *Lancet* for July, 1879, with the addition of some further cases and details. It presents a careful summary of our present knowledge of the clinical history and pathology of this disease, to which the author has made valuable contributions. Dr. Gowers believes that the change in the muscles is a morbid growth of connective tissue and fat in the place of striped muscular fibre, and he regards the condition as essentially a perversion of development. The changes in the spinal cord which he and the late Dr. Lockhart Clarke described were slight, and he believes were probably secondary, as many com-

petent investigators have in other cases found the cord normal; where more definite changes—e. g. sclerosis of lateral columns, atrophy of nerve cells—have been found, he thinks there is reason for believing that some other pathological state has been confounded with this disease. In detailing the clinical history, which is founded upon an analysis of 220 cases, 19 of which have come under his own observation, Dr. Gowers directs attention to the distinctly hereditary nature of the affection, in some instances six or even eight members of the same family having suffered from it; to its tendency to affect males rather than females, in the proportion of 6 to 1; to its incidence at an early age, usually before six years. He describes the insidious manner in which the disease reaches development; weakness of the knees, and slight unsteadiness of gait being often for some time the only facts observed, and as often little regarded. The *enlargement* of the muscles, which has been so much insisted upon, may be absent, and affects as a rule only certain groups, notably those of the calf, the infra spinati, deltoids, and triceps; *wasting* of the muscles is quite as common, but affects different groups, especially the latissimi dorsi and pectorals. But both enlarged and diminished muscles are weak, the symptoms of the disease are the result of this weakness, and Dr. Gowers lays great stress upon the efforts made by the little patients to raise themselves from the ground, by grasping their knees with their hands as a peculiar movement quite pathognomonic in its significance. The treatment recommended is chiefly to improve the general nutrition by arsenic and cod-liver oil, and the muscular nutrition by faradism and moderate gymnastic exercise, aided by friction; but unfortunately little can be hoped for, as scarcely an authenticated cure has been recorded, the patients becoming emaciated, their respiratory muscles failing, and death finally resulting from some acute or chronic pulmonary disease.

Dr. Gowers has increased the value of his work by an appendix giving notes referring to several points of interest, by a copious bibliography, and by a lithographed plate of the microscopical appearances of the diseased muscles.

ROBERT SAUNDBY, M.D.

The Index Medicus. A Monthly Classified Record of the Current Medical Literature of the World. Compiled under the supervision of Dr. John S. Billings and Dr. Robert Fletcher. New York, F. Sypfyoldt. London, Trübner & Co.

It is impossible to exaggerate the utility of this work, or to extol too highly the accurate industry of those who are engaged in its compilation. Recording as it does all new publications in medicine, surgery, and the collateral branches of science, and all original communications in medical journals, and transactions of medical societies, it arranges these in a manner that renders reference easy, the nomenclature and classification adopted being those of the Royal College of Physicians of London, based on Dr. Farre's well-known system. It brings compendiously before the worker in each department all contemporaneous research and speculation in his own field; it catalogues all substantial additions to medical knowledge, and it provides an inventory of the passing medical fashions of the day. To all medical practitioners, teachers, and authors it must prove useful, but to the provincial student, who has not access to medical libraries and their array of journals, it will be especially valuable, by enabling him to ascertain what is being done by others in any subject that he may be investigating, thus guiding and stimulating his explorations, and saving him from the repetition of twice-told tales. Happily provincial students of medicine—that is to say, those medical men who are not content with the routine of practice and money-making, but who feel the obligation under which they lie to promote the progress of medicine—are daily increasing in number. The country districts furnish much admirable work, and we think that the 'Index Medicus' will tend to improve the quality of this work and increase its quantity, while at the same time it diminishes its bulk.

J. Crichton-Browne, M.D.

Skin Diseases, including their Definition, Symptoms, Diagnosis, Prognosis, Morbid Anatomy and Treatment. A Manual for Students and Practitioners. By MALCOLM MORRIS. London. Smith, Elder, & Co., 1879. 8vo, pp. 288.

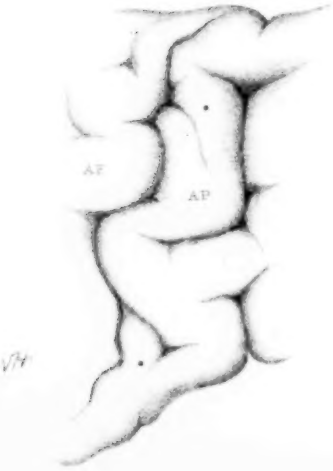
THE author of this manual has evidently a full and intimate acquaintance with the literature of dermatology, and with the most recent developments and appliances of cutaneous medicine. He has produced a plain practical book, by aid of which, who so chooses, may train his eye to the recognition of slight but significant differences. The descriptions are neither too vague nor over-refined; the directions for treatment are clear and succinct. We cannot add, however, that Mr. Morris has surmounted the everlasting stumbling-block of classification, his system being compounded out of those of Erasmus Wilson and Hebra, with original touches here and there. Under the class Neuroses we find zoster, cheiro-pempholyx, pruritus, and dystrophia cutis, while urticaria takes place under the Erythematous Exudationes, pemphigus under the Vesicular Exudationes, morphœa under the Hypertrophiæ and alopecia areata under Diseases of the Hair. An appendix of formulæ for baths, ointments, lotions, &c., would much enhance the value of the book.

J. CRICHTON-BROWNE, M.D.

Fig. 1. (Right)

Fig. 2. (Left.)

Fig. 3



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Clinical Cases.

ARREST OF DEVELOPMENT IN THE LEFT UPPER LIMB, IN ASSOCIATION WITH AN EXTREMELY SMALL RIGHT ASCENDING PARIETAL CONVOLUTION.

BY H. CHARLTON BASTIAN, M.D., F.R.S.,

and

V. HORSLEY,

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In the number of 'Brain' for October 1878, Dr. Gowers has published an account of a case in which congenital absence of the left hand co-existed with unequal development of the ascending parietal convolutions, the right being the smaller. Towards the close of 1879, a case, which has since proved to be very similar, was admitted into University College Hospital, under the care of Dr. Bastian. The following facts concerning this case seem to us worthy of being recorded.

J. M. B., aged 26, a clerk, was admitted into the hospital on October 18th, 1879, suffering chiefly from mitral obstruction and regurgitation. He got rapidly worse (owing in part to rupture of one of the chordæ tendineæ of the mitral valve), and died rather suddenly on October 30th, 1879.

Necropsy, Oct. 31*st*, 1879. The body, on the whole, showed slight muscular development, especially in and in relation with the left upper limb, both pectoral muscles on that side being absent. Unfortunately, permission to dissect the limb (which was shorter and much slighter than that of the right side) could not be obtained, but the arrangement of the bones in the small and abortive "hand" was readily felt through the skin, and is represented in the accompanying figure (H), copied from a sketch made by the physician's assistant, Mr. B. Pollard. The dotted lines show the outlines of the bones. The position and proportion of the metacarpals bear a close resemblance to the early appearances of segmentation, seen as slight ridges and intervening depressions in the forelimb of an embryo (man)

about 2 cm. in length. In the case of Dr. Gowers, above referred to, in which one of us had the opportunity of dissecting the limb, the theory of intra-uterine amputation was favoured by the condition of the parts; but in the present instance the appearances are strongly in favour of arrest of development.

The brain, on removal, was divided longitudinally, and each half placed on its inner surface in a 2·5 per cent. solution of bichromate of ammonia. The membranes were at once removed over the ascending convolutions on each side, when a striking difference appeared (as shown in figs. 1, 2). After 24 hours, the middle three-fifths of each ascending parietal convolution was measured at seven equidistant points between the dots in each figure, and the following results were obtained:

	Left A.P.C.	Right A.P.C.
	cm.	cm.
1	1·4	1·5
2	1·6	1·5
3	1·5	1·85
4	1·4	1·7
5	1·3	·8
6	1·9	1·0
7	1·8	1·3

Thus it is seen that at the points 5 and 6, the difference in breadth was almost 2 to 1 in favour of the left asc. par. conv., and that, on the whole, there was a difference of ·2 cm. between the right and left convolutions.[1] There was no such striking asymmetry in the rest of the brain; the basal ganglia and descending motor tracts in the pons and medulla being apparently equal. A careful microscopical examination revealed no appreciable difference in the internal or minute structure of the two convolutions.

From the above facts we see that the regions of the cortex, corresponding with the "centres" "*a*, *b*, *c*, *d*" of Dr. Ferrier,[2] were but slightly developed on the right side. The actions evoked by stimulation of these centres he gives as, "Individual and combined movements of the fingers and wrist, ending in clenching of the fist." And in this man such movements had certainly been for the most part impossible with the left "hand."

It would seem, then, that we have here another instance tending to corroborate the view that there is a correlation of some kind between the functional activities of these regions of

[1] The drawings were obtained by taking impressions of the sulci by means of tinfoil. The deepest portions of the sulci being marked by punctures, the tinfoil was laid on the paper, and the latter marked through the holes. The accuracy of the drawings was finally verified by numerous comparative measurements. The actual breadth of the convolutions themselves was filled in from the above measurements, and the figures were then reduced one-third.

[2] 'Functions of the Brain,' pp. 148 and 307.

the ascending parietal convolution, and the movements of the opposite hand and fingers—as indicated by the experimental observations of Dr. Ferrier.

It seems something more than can be accounted for by mere chance coincidence, when precisely the regions of the cortex indicated by him are found to be defective in bulk in two consecutive cases of absence or arrest of development of the hand. Yet those who are still a little sceptical will doubtless await further coincidences of the same kind, and will seek to learn something definite as to the degree of frequency with which a marked asymmetry of the ascending parietal convolutions of the two sides is to be met with in cases where no malformation of one hand is present.

The question of the existence or not of a correlation of this kind is one which does not by any means necessarily associate itself with the view that the particular regions of the cortex indicated contain "motor centres." A correlation may really exist between the activities of special cortical regions and the performance of particular movements, and yet the commonly received explanation of such a correlation may not be correct.

Thus it is possible to accept as a fact the existence of such correlations, and at the same time wholly to reject the view that anything like a "motor centre" is to be found in the cerebral cortex.

A complex group of ingoing impressions follows or is produced by movements of the hands, as of all other parts of the body, so that we may be said to have a "sense of movement" growing out of the repetition of what one of us has termed kinæsthetic impressions[1]—just as our sense of sight is developed from the repetition of visual impressions.

Sensory impressions of both these types, derived from movements, will subsequently, when ideally revived, co-operate as factors in the reproduction of similar movements. The activity of the "kinæsthetic centres," more especially, would thus be closely related to the reinitiation of movements—and different parts of such centres would have to do with the production of different kinds of movements. Thus stimulation of certain sensory regions of this type may lead to the manifestation of certain movements, whilst destruction of the same sensory regions may abolish the possibility of performing such movements. The so-called "motor-centres" may, therefore, in reality, be sensory regions of the kinæsthetic type, and these cortical areas may in addition take part in the performance of other cerebral functions whose nature cannot be at present specified.

[1] 'The Brain as an Organ of Mind' (1880), pp. 543 and 522.

The fact that the small right ascending parietal did not differ in its microscopical characters from the well-developed left ascending parietal convolution, is not incompatible with the existence of a correlation of some kind between these parts and certain movements of the hand, whether their activities be of the sensory or of the motor type. In such a case as we have now brought forward, there would be no reason to look for atrophied nerve cells, merely because certain movements have never occurred, but rather for a numerical inferiority of nerve cells and a mere inferior bulk of convolution in relation with an abortive limb whose possible movements were notably curtailed.

CASE OF NERVE-SPLITTING.

BY SURGEON-MAJOR K. M'LEOD, A.M., M.D., F.C.U.,

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C. R., æt. 26, a spare East Indian youth of nervous temperament, consulted me on the 27th of December regarding a wasting of the left forearm and hand, and numbness of the latter, from which he had suffered for 8 years, and for which he had been subjected to various plans of treatment, without gaining relief from any. In the year 1871 he began to experience peculiar sensations in the little finger and ulnar side of the left hand. The parts gradually got numb, the muscles of the hand wasted, and the fingers became permanently bent. He sought relief at the Presidency General Hospital, when it was discovered that the ulnar nerve had undergone thickening. Blisters were applied along the course of the nerve, and subsequently iodine and magnetic electricity, but no benefit was derived from these energetic remedies. An abscess formed in 1875 above the elbow on the inner side, which discharged spontaneously, and left a sinus which continued to emit matter for some time, and then closed of itself. This sinus reopened in 1879, without apparently any fresh accession of inflammation. His condition when I examined him was as follows:—

Left arm generally less muscular than the right; circumference of left forearm 1 inch less than right. Special wasting over position of flexor carpi ulnaris. Hand very much wasted, more particularly the short muscles of the little finger, the interossei, and the adductor pollicis. The short muscles of the thumb are smaller in bulk than on the opposite side. The fingers are habitually bent, more especially the ring and little fingers, which are permanently flexed at both phalangeal joints. The thumb can be straightened completely, the fore and middle fingers almost completely, the ring and little fingers very partially, the first phalangeal joint remaining bent at a right angle. The action of the common extensors and flexors is unimpaired. The power of adduction and abduction of the fingers is almost

abolished. The thumb can be adducted and abducted. Sensation is abolished all over the little finger, and over its metacarpal bone on the dorsal and palmar aspect as far as the styloid process of the ulna. Sensation on the ulnar side of the ring-finger is diminished, and on the radial side perfect. Sensation over the ulnar two-thirds of the dorsum of the hand (as far as the line of the metacarpal bone of the middle finger) is very much impaired. On the palmar side sensibility extends as far as the metacarpal bone of the ring-finger. The degree of sensibility was tested by pricking with a pin, and the patient, who is a very intelligent young man, gave very prompt and definite indications of the sensations experienced.

The skin of the insensible area is somewhat congested, but there is no vesication or breach of surface. There is a hard knot on the dorsal branch of the radial nerve about 3 inches above the wrist. There is a similar knot on the great auricular nerve of the left side as it crosses the sterno-cleido-mastoid muscle, and on the frontal nerve of the right side. All these knots are painful on pressure. The ulnar nerve is very much thickened behind and above the elbow to the extent of about 5 inches. It is hard and cartilaginous to the feel, as thick as the middle finger, and painful on manipulation. There is an orifice of a sinus over the course of the nerve situated about 4 inches above the inner condyle of the humerus.

I advised the patient to submit to the operation of stretching the ulnar nerve as the only expedient likely to benefit him, and gained his ready consent.

Operation.—After bringing him under the full influence of chloroform, and washing the parts thoroughly with carbolic lotion, I passed a director into the sinus, and found that it passed into the interior of the nerve as far as the level of the inner condyle. I slit the sinus up throughout its whole extent. A long linear incision, exposing the nerve for a space of 4 inches, was the result. It was hollowed out by an abscess of which its thickened texture constituted the wall. This cavity was filled with curdy material, and stuff of the same kind escaped on pressure from orifices in the abscess wall. These orifices led to small chambers or recesses in the mass of the thickened nerve. The cavity was thoroughly emptied, and the contents of the flask-shaped recesses squeezed out. The lining membrane of the main cavity was then scraped off, and the smaller cavities carefully cleaned out with a small scoop. The nerve was then split in two from before backwards, and the division was prolonged into the comparatively sound nerve above and below, the knife following as far as possible the direction of the fibres. The continuation of the nerve behind the condyle was somewhat thickened, and contained a few cells

full of yellow curdy material. The operation was performed under strict antiseptic precautions, and the whole line of wound was very carefully brought together by iron wire and horse-hair stitches, after a few threads of carbolised catgut had been laid in it for drainage. Boracic-acid ointment, spread on muslin, was applied next the wound, and the ordinary carbolic gauze-dressing placed so as to reach to the axilla above and half-way down the forearm below. The dressing was removed every second or third day, according to circumstances. Convalescence was satisfactory. An attack of ague occurred during the second day, which yielded to quinine. The neighbourhood of the wound was intensely sensitive for the first ten days or so. The wound remained sweet and healed kindly. The following notes were taken on the 21st of January, 1880—24 days after the performance of the operation :—

"Cicatrix completely healed. No abnormal sensibility in the neighbourhood of the wound. The thickened nerve is still perceptible beneath the cicatrix, but it is not so hard or bulky as before the operation. There is no evidence of deep matter nor sinus. Above the cicatrix the nerve is slightly thickened and very sensitive; below the cicatrix the nerve is also somewhat thickened and sensitive, but less so than above. The flexor carpi ulnaris is still atrophied. He straightens his little and ring-fingers rather better, and separates his fingers with more force and effect. The muscles of the hand are still very much wasted, and the tendons very prominent. There is still slight congestion of the dorsal surface of the ungual phalanx of the little finger. Painful sensation is elicited by the prick of a pin all over the hand, except over a narrow strip on the dorsum of the little finger, as far as within half an inch of the styloid process. Pinching with the finger and thumb, however, produces pain up to the first phalangeal joint; beyond that there is no sensation, or perception of pricking, or pinching, but when the end of the finger is forcibly squeezed, pain is caused."

Remarks.—The condition for which the operation above described was resorted to is, as far as my experience goes, a very rare one. I have frequently seen thickened nerves leading to anæsthesia and impaired motility over the area supplied by them; but I have never before seen a nerve hollowed out in this fashion by an abscess, and the seat of curdy deposits. In this patient three nerves were the subject of thickening, and two of them of abscess and sinus. The ulnar nerve appears to be specially liable to this thickening. Dr. Laurie, acting on a suggestion which I threw out in 1877, stretched this nerve in 30 cases in which thickening and anæsthesia existed ('Indian

Medical Gazette,' vol. xiii., pages 229, 270), and in all of which the operation produced benefit. In two cases, regarding which he has been able to obtain information, marked and permanent improvement was experienced; and in a similar case, which I reported at length in the August (1879) number of the same journal, restoration of sensibility and power followed very speedily upon the stretching of the thickened nerve. In the present instance stretching was not resorted to—simply laying open and splitting the nerve—and although the patient did not recover sensibility or muscular power fully, the notes of the condition before and after the operation leave no doubt that very considerable amelioration was caused by the operation in both respects. How the operation of mechanical stretching restores the function of the nerve I am not prepared to say. The case now recorded would render it probable that amendment is produced by removing tension and pressure upon the nerve fibrils. The case is also interesting in demonstrating the function of the interossei and lumbricales muscles in extending the two terminal phalanges. Their tendons join the extensor aponeurosis on the dorsal surface of these phalanges. There was no impairment of power of the common or special extensors, but the paralysis of the interossei and lumbricales caused permanent flexion of all the fingers, and when the function of the ulnar nerve was partially restored, the fingers could be better straightened.

CASES OF INTRACRANIAL TUMOUR.

BY JAMES ROSS, M.D.,

Assistant-Physician, Manchester Royal Infirmary.

CASE II.—Samuel Holmes, æt. 7 years, presented himself as an out-patient at the Southern Hospital, Manchester, on January 26th, 1876.

The following history was elicited from the mother:—He was a bright, intelligent, and healthy boy until about 15 months ago, when he fell from a wall, 5 feet high, and raised a lump on his forehead. Soon afterwards he complained of constant headache, chiefly confined to the forehead. The top of the head was also so sensitive that combing his hair caused him much pain. He could not keep still; his legs, especially, were constantly moving, and at meal times he was in the habit of knocking the table with his right hand, as if from impatience. About nine months ago the mother noticed that his mouth was slightly "crooked," and that his left arm hung helplessly by his side. The forearm was twisted so that the palm of the hand was directed outwards and the thumb backwards, his fingers were bent, but she thinks his thumb at first was held straight and drawn away from the fingers. After some weeks, however, the thumb became bent inwards under the index-finger, and she had to pare the nail of the thumb frequently to prevent its cutting the skin of the outside of the middle finger. He now began to drag the left foot in walking, and the forearm was gradually drawn up behind his back, instead of hanging, as at first, by his side.

The mother had nine of a family, no miscarriages and no still-born children. One child died from convulsions during teething; a second child, who was weakly from birth, died at three months of age; and a daughter has suffered for the last two years from white swelling of the knee.

On presenting himself at the hospital he was a well-made and fully-developed boy for his years. His head was large, but well-proportioned; face round and plump, although pallid; his incisor teeth were regular, his nose was well formed, the

muscular system was well developed, and there was abundance of subcutaneous fat. There was very well-marked left facial paralysis, so that the left corner of the mouth could not be moved in the slightest. Both eyes could be closed; the pupils were large, equal, sensitive to light, and there was no affection of the special senses. The left elbow was kept a little behind the mesial plane, and 2 inches from the side; the forearm was bent at right angles to the arm, and drawn behind the body; the hand was strongly pronated; the thumb was adducted, and the second phalanx flexed, so that the point rested against the second phalanx of the middle finger. The first phalanges of the fingers were extended and in a line with the metacarpal bones, and the second and third phalanges were flexed. A considerable amount of muscular rigidity was induced on attempting passive motion at the elbow and wrist joints. By a voluntary effort he could raise his elbow to nearly the level of the shoulder, and bring the upper arm slowly forwards; but he could neither extend the forearm, produce supination, nor extend the fingers. The left leg dragged during walking, but there was no muscular rigidity, and all the movements of the leg could be separately performed. The electro-cutaneous sensibility of the left half of the body was increased, especially over the back of the left hand, and the left half of the face and side of the head. The slightest touch of the skin over the vertex of the head to the left of the middle line caused the patient to wince, and the cutaneous sensibility to pain was increased over the left half of the body generally. The other organs were healthy, and there was no albumen or sugar in the urine. He was ordered four grains of iodide of potassium three times a day; but, as no improvement took place, he was admitted into the hospital on February 28th.

March 10th, 1877.—He was ordered, on admission, fifteen minims of the syrup of the iodide of iron, to be taken three times a day, and the daily application of a weak constant current to the paralysed muscles and nerves. After two applications of the constant current he could extend his fingers to a slight extent, and in a few days he was able to raise his hand to the back of his head. It was observed, however, that the most marked improvement took place at the shoulder-joint; and that improvement in the movements of the forearm and hand was only to a slight extent. This improvement was of short duration, and he now looks decidedly worse than on admission. The pallor of the face is much increased; his appetite has failed; the pulse is 110, weak and irregular; and the nurse says that he has become very stupid. Ordered to be kept in bed, milk diet and a saline mixture.

March 18th.—Since last report he has got steadily worse, has

vomited frequently, and to-day he has been seized with general convulsions. The convulsions frequently occurred for the next two days, and he did not recover consciousness in the intervals, and died early on March 21st.

Sectio cadaveris, twelve hours after death.—On opening the skull, the convolutions of the brain presented a flattened and compressed appearance, and about 2 ounces of fluid escaped during removal. The brain weighed 51 ounces. On slicing the brain to a level with the corpus callosum the upper surface of a tumour was exposed, which was situated in the centrum ovale of the right hemisphere, immediately to the right of the corpus callosum and at the junction of the anterior and middle lobes. On opening the lateral ventricles, this tumour was felt as a hard nodule, slightly projecting into the right lateral ventricle, and occupying the position of the caudate nucleus and anterior portion of the optic thalamus, and only covered by the ependyma of the ventricle. The tumour measured three-quarters of an inch in the transverse and an inch in the antero-posterior and vertical diameters respectively, so that not only the caudate nucleus and anterior portion of the optic thalamus, but also the anterior two-thirds of the internal capsule and the anterior portion of the lenticular nucleus were destroyed by it.

The growth was pretty sharply defined from the surrounding brain-tissue, and on section it presented an outer grey, somewhat vascular cortex, about two lines thick, and a central core of a yellow colour, and apparently destitute of any structure.

Microscopic examination showed that the grey cortex of the tumour consisted of giant cells, each surrounded by lymphoid cells imbedded in a fibrillated reticulum.

The right lung was closely adherent to the chest wall and to the diaphragm. The lung itself was congested, but every portion of it floated in water. No tubercles nor cheesy glands were discovered, and the other organs were healthy.

Remarks.—The progressive character of the symptoms in this case, as well as the fact that its origin dated from a fall on the head, pointed from the first to the growth of an intracranial tumour. My diagnosis, therefore, was tumour, probably tubercular, of the right corpus striatum. At that time I was not aware of the full significance of the fibres which pass through the internal capsule to reach the cortex of the brain. Looking back on the case now, I think it probable that the tumour began first to grow from one of the arteries which branch from the middle cerebral artery and pass through the anterior perforated space to reach the caudate nucleus of the corpus striatum. It has occurred to me that the thumping

movement of the right hand described by the mother might really have taken place in the left hand, and that these, along with the other restless movements, might have been due to primary irritation of the left caudate nucleus or the *nodus cursiosus* of Nothnagel. This supposition, however, is too doubtful for any reliance to be placed upon it. The paralysis is to be explained on the supposition that the fibres of the middle third of the internal capsule, and which connect the cortex of the brain with the spinal cord, were gradually compressed by the progressively increasing size of the tumour. These fibres were therefore compressed from before backwards, and it is important to notice that the left half of the face was more paralysed than the upper extremity, and the upper much more paralysed than the lower extremity.

The peculiar position occupied by the forearm and hand is also interesting in connection with the gradual compression of the motor tract in its passage through the internal capsule. The sensory disturbances were probably due to irritation of the fibres of the posterior third of the internal capsule. Veyssière has proved that section of these fibres in dogs is followed by hemianæsthesia of the opposite side, and the cases recorded by Dr. Hughlings-Jackson and others show that a lesion of the posterior portion of the optic thalamus, involving the posterior fibres of the internal capsule and the external geniculate body, is attended by hemi-anæsthesia of the opposite side along with hemiopia. It may, therefore, be inferred that irritation of these fibres will cause hemi-hyperæsthesia of the opposite side.

CASE OF ALTERNATE HEMIPLEGIA, WITH CONJUGATE DEVIATION OF THE EYES.

BY ALEXANDER M. McALDOWIE, M.D.,

Resident Medical Officer, North Staffordshire Infirmary.

In March 1876 I was sent for to see John S., aged 29, a labourer, stated to be suffering from a "stroke of paralysis."

The following history was elicited:—

The patient contracted syphilis eight years ago, and suffered from ulcerated throat and skin eruptions for several years subsequent to the chancre, but has been free from these complaints for more than two years. Has been troubled with pains in the shins and arms for nearly six months, and from dull aching pains in the occipital region, together with giddiness, almost every night for the past three months. Four days ago the patient suddenly lost the use of the right arm and leg while dressing himself in the morning. There was no loss of consciousness at the time of the attack, although the patient felt giddy and his eyesight grew dim. The power of speech was lost for some hours. Patient had felt no premonitory symptoms.

Present condition.—Patient well nourished. Intellect very good. Complains of severe pain in the occipital region. Pain heavy and dull in character, most intense during the night. Total paralysis of the upper, and almost entire loss of power over the lower, extremity on the right side. (Patient states that the paralysis of the leg was complete for two days after the attack.) Sensation greatly diminished in the paralysed limbs; electrical excitability impaired; sensation of heat and cold normal. Complete paralysis of the left side of the face. Tongue protruded straight. Speech thick and very indistinct, but no aphasia. Ptosis of left eyelid; slight twitching of right upper eyelid. Marked deviation of both eyes to the right, the patient being unable to turn them towards the left beyond the middle line. Pupils natural in size, react readily to stimulus

of light. Ophthalmoscopic appearances normal, both discs pale, outline well defined, vessels natural. Nothing abnormal detected on examination of the chest and abdomen.

Ordered 10 grs. of iodide of potassium three times a day.

The patient improved steadily under treatment. The deviation of the eyes disappeared about three weeks after the attack, but the facial paralysis lasted for nearly two months. He regained the power of the leg so as to be able to walk fairly well. The arm remained very weak, the patient being able to use the hand and forearm, but was unable to raise the limb to the horizontal position.

The treatment was anti-syphilitic throughout, iodide of potassium being administered at first, afterwards liquor hydrargyri perchloridi, and finally biniodide of mercury.

The patient was lost sight of about four months after the attack.

Remarks.—A case of hemiplegia alternans with conjugate deviation of the eyes, is of considerable interest and worthy of record.

Alternate hemiplegia is the most characteristic symptom of disease of the lower part of one lateral region of the pons. In this case the lesion was evidently situated at the lower part of the left side, involving the fibres of the facial nerve below their decussation in the pons, and the motor fibres for the limbs above their decussation in the medulla.

The completeness of the facial paralysis observed in this case is also a prominent sign of disease in this region. Bastian says that in lesions in this part "we have an unusually well-marked facial paralysis on the side of the brain lesion, and a more or less complete motor and sensory paralysis of the limbs of the opposite side." And Nothnagel observes: "In this variety of facial paralysis of cerebral origin, all the branches of the facial nerve, even those supplying the frontalis, corrugator supercilii, and orbicularis palpebrarum, are involved, just as in the peripheral form of this affection; whereas, when the lesion is situated in the upper part of the pons, at least in the cases which I have observed, these branches escape."

The conjugate deviation of the eyes from the side of the brain lesion and towards the paralysed limbs noted in the above case, also points to a lesion of the pons. Prévost was the first to point out that the deviation of the eyes follows a constant law, being always towards the side of the brain lesion. The only exceptions that he admits are affections of the pons and medulla, when the deviation may be away from the side of the brain affected. Bernhardt argues that this symptom has

no diagnostic value, but the small number of exceptions that he brings forward only show that the law is not so constant as Prévost maintains. Taken in connection with the alternate hemiplegia observed in this case, the symptom is of importance in aiding us to localise the disease.

As to the nature of the affection, judging from the history and the result of the treatment adopted, there can be no doubt but that the disease was of syphilitic origin, probably the first form of "syphilitic new-growth" described by Heubner.

CRURAL MONOPLEGIA—LIMITED CORTICAL LESION OF OPPOSITE HEMISPHERE.

BY DAVID FERRIER, M.D., F.R.S.

King's College Hospital, and National Hospital for the Paralysed and Epileptic.

On November 18th, 1879, I saw S. W., ætat. 40, of whose case the following are chief particulars, as furnished me by Dr. Brookhouse, of New Cross, with whom I saw the patient in consultation.

The patient was first seen by him on Sept. 21st, 1879, and had been feeling "out of sorts" for a week previously with symptoms of what he called a bad cold. He had a temperature of 101° F., pulse 100, anxious expression, and some abdominal tenderness, but no diarrhœa. He had a slight cough, but there were no abnormal physical signs on auscultation and percussion.

His symptoms fluctuated between this time and October 9th, but he became weaker; his appetite became impaired, his tongue coated and flabby, and his breathing became laboured on exertion. Nothing could be detected on careful examination of the lungs except a few moist râles at the right base posteriorly. The symptoms in many respects resembled typhoid.

On Oct. 24th he complained of some stiffness in the left leg, and inability to move it so freely as the right. On the 25th it became still more feeble, and on the 26th it became completely paralysed as to motion.

This continued till Oct. 28th, when he said that he feared the left arm was "going like the leg." The grasp of the left hand was distinctly feebler than the right. Next day the feebleness was still more pronounced, and by the 31st the left arm had become completely paralysed like the leg.

During the night of Nov. 6th, he had a series of convulsive movements of the left leg and arm, commencing in the leg and lasting half an hour.

From this date the patient grew worse. His temperature

ranged between 99° and 102°·4 F., there being no marked difference between morning and evening temperatures. There was no dulness in the lungs, but fine crepitation on both sides posteriorly. There was much cough, with frothy yellowish spatum, but quite free from any rusty appearance.

On Nov. 18th I saw the patient with Dr. Brookhouse with a special view to determine the nature of the paralytic affection. The patient was somewhat flushed, breathing rapidly and with an appearance of oppression when he spoke, coughing a good deal and expectorating frothy mucus. His temperature taken on the right axilla was 102° F., while on the left axilla it was 102°·8 F.

There were no symptoms of extensive cerebral disease. The mind was clear. There was no headache or vomiting, and examination of the discs revealed nothing abnormal.

There was nothing abnormal discoverable on examination of the abdomen. The urine was free from albumen. The lungs were resonant throughout, but there were moist sounds at the base posteriorly, particularly on right side. On examination of the limbs the left leg and left arm were seen to be perfectly flaccid and powerless. There was a total loss of volitional power. There was no trace of facial paralysis. Both sides acted equally well, and the tongue was protruded straight.

There was increased tendon reflex in the left leg. Sensasation was unimpaired. The slightest touch on any part of the left arm or leg was distinctly perceived and accurately localised.

The patient had had syphilis and had lately been under specific treatment, owing to suspicious syphilitic ulceration of the tongue. A sister had died of acute tuberculosis. From a consideration of the past history and symptoms presented by the patient, I formed the opinion that the constitutional symptoms and febrile excitement depended mainly on acute infiltration of the lungs, either tubercular or syphilitic, and that the paralysis depended on a limited lesion of the cortex, progressive in character, and situated at the upper extremity of the fissure of Rolando on the right hemisphere.

An unfavourable prognosis was pronounced.

No specially fresh symptoms occurred, but the patient gradually sank and died on Nov. 24th, in a state of coma.

Post-mortem examination.—On November 26th we made a post-mortem examination, but had to confine our examination to the head and partial exploration of the thorax.

The skull-cap was easily detached and was free from adhesions. The dura mater was also easily separable, except for slight, easily broken down, adhesion at the middle line at

a region corresponding to the upper extremity of the fissure of Rolando on the right side. The subarachnoid space contained some fluid, but clear and free from opacity. The pia mater was everywhere normal and separated readily from the cortex except at one spot. This was situated at the upper margin and internal aspect of the right hemisphere on both sides of the fissure of Rolando, where the pia mater merged into a caseous adhesion not appreciably elevated above the rest of the cortex, but which could not be removed without tearing the cortical substance, and causing erosion.

On further careful examination of the position and extent of the lesion, the following notes were made:—

The pia mater was adherent and studded with minute tubercular foci, of a yellowish aspect, on the internal aspect of the hemisphere at the point where the fissure of Rolando terminates. The adhesion was of a quadrilateral shape, measuring 1 in. antero-posteriorly and $\frac{3}{4}$ in. vertically. (The position and extent of the lesion here will easily be understood by reference to Ecker's figure of the internal aspect of the hemisphere. It occupied an area which would be included between the letters A and B, and concealing C.)

Doubling over the margin of the hemisphere the adhesion separated the extreme upper lips of the fissure of Rolando from each other, and descended along the bottom of this fissure, coming to a point an inch below the margin of the hemisphere. The whole area and shape of the lesion may be likened to a triangle, the base of which would measure 1 inch, and the height $1\frac{3}{4}$ inch or 2 inches at the utmost, applied with its base at the internal termination of the fissure of Rolando, and doubled over the margin with its apex directed along the bottom of this sulcus.

Removal of the adherent membrane caused tearing and erosion of the cortex, but quite superficially; the medullary substance underneath being injected but not softened.

The degeneration was most advanced internally and at the extreme upper margin of the hemisphere, as indicated by the caseous softening of the miliary tubercles.

There was no excess of fluid in the ventricles, and the rest of the brain was normal throughout.

The lungs, portions of which were removed and examined microscopically were studded throughout with miliary tubercles, feeling to the touch as if infiltrated with small shot. There were no cavities or areas of softening.

The other viscera were not examined.

Remarks.—This is an important addition to the comparatively small number of clinical cases serving to indicate the

position of the leg-centre in the cortex. It is an altogether uncomplicated instance of purely cortical lesion, so definite as almost to compare with a physiological experiment. Though the paralysis did not remain confined to the leg throughout, yet the fact that it did so for four days clearly proves that the leg-centre is capable of differentiation from those of the arm, though they are so commonly implicated together. The point where the lesion began is indicated by the region where the caseous degeneration was most advanced. This was the inner aspect and margin of the hemisphere on each side of the upper extremity of the fissure of Rolando. This situation accurately corresponds to the areas—1 and 2—which I have indicated on the monkey's brain as comprising the centres for the various movements of the leg. The extension of the paralysis to the arm is in accordance with the advance of the lesion along the fissure of Rolando.

The diagnosis—which I made with confidence from the mode of onset and its progress, the character of the paralysis, and absence of general cerebral symptoms—of a limited cortical lesion of a progressive character, situated primarily at the upper extremity of the fissure of Rolando, was completely verified by post-mortem examination. The case shows *inter alia* that with lesions strictly cortical there may be the most absolute motor paralysis, with perfectly intact cutaneous sensibility; and, further, that with cortical lesions vaso-motor paralysis is associated with motor paralysis, as indicated by the higher temperature—·8° F.—of the paralysed side.

Abstracts of British and Foreign Journals.

Innervation des Vaisseaux cutanés, Dastre et Morat. (*Archives de Physiologie*, Mai—Août, 1879.)—The above paper enters into an exhaustive critique of vaso-dilatation, and furnishes certain precise data towards the solution of its problem. The central origin of vaso-constrictor nerves is first discussed—it is now-a-days proven that they are associated with the anterior spinal roots, the dorsal division of the cord being their principal focus of origin; but there is a further centralisation of influence within a limited portion of the bulb, the general tonic centre or vaso-motor centre of Owsjannikow; and vaso-motor centres have further been assigned even in the cortex. Of the degree of centralisation and of the relative independence of an axial series of spinal centres, D. and M. remark that the universal influence of the bulbar centre is undeniable, but that this influence is not exclusive—the conclusion is that vaso-constrictor activity has one chief source (bulbar) and several accessory sources (spinal and encephalic). Still another element comes into account—the possible or probable existence of peripheral tonic centres (Huizinga) constituted by the ganglionic cells in intrinsic plexuses of the arterial wall—intrinsic arterial ganglia. One among many good arguments in their favour is as follows: a dilatator (e. g. corda tympani) contraction of the vessels by irritation of the sympathetic is hindered by simultaneous irritation of the corda; since the nerves run independently to the periphery, it is here that we must presume some mechanism to exist compounding their opposite actions. The question of dilatation is considered at length—whether it is by an activity opposed to constriction, or merely by an absence of constriction.

The definition of a dilatator nerve is insisted upon as a centrifugal nerve whose excitation effects a primary dilatation of vessels independent of any central mediation, thus excluding the confusing consideration of reflex dilatations. We are agreed as to the mecha-

nism of constriction, not so as to that of dilatation. Dismissing the notions of a direct dilatator influence of the nerve fibre on the muscle fibre, or of the dilatation of arterioles by constriction of venules, or of exaggerated peristaltis, it cannot be denied that every dilatation is a paresis of constriction, and before the suggestion of dilatators this was the universal statement; but the discovery of the dilatator action of the corda tympani led to the generalisation of active dilatation opposed to contraction, and subserved by vaso-dilatators distributed with vaso-constrictors throughout the body. Dilatators were then sought for, and under favourable circumstances for the most part found. The sciatic has been throughout a chosen bone of contention; the notorious discrepancies of results are attributed by D. and M. to inadequacy of tests. The most employed has been thermometry; less frequently, observation of a bleeding-point and plethysmography. The thermometer is unsatisfactory, because it is a sluggish test, and because it is not proved that temperature varies coincidently with dilatation; note also that in the scale of low temperatures, with constriction of vessels, dilatation is most readily manifested and its thermic effect greatest, and *vice versâ* in the scale of high temperatures; further that when vessels dilate warmth increases, and dilatation is therefore cumulative. Classing observers into: (1) those to whom the sciatic is a dilatator (Golz, Masius, Vanlair); (2) those to whom it is constrictor *and* dilatator, according to circumstances (Onimus, Lépine, Kendall and Luchsinger, Bernstein and Marchand, Grützner and Heidenhain); (3) those to whom it is a constrictor (Putzeys, Tarchanoff, Vulpian)—D. and M. remark that classes (1) and (2) employ the thermic test, class (3) have recourse to direct observation.

D. and M. resorted to manometry.—They first estimated the local variations of pressure effected by an accepted constrictor (sympathetic), with these they then compared the local variations effected by a mixed nerve (plantar branch of sciatic). The results were in either case identical. With the sympathetic, facial vein and facial artery of horses or asses, they obtained with the section of symp. (1) a brief elevation of both pressures (lasting 6″ to 8″), due to constriction of vessels by the momentary irritation of section, followed by (2) a gradual rise of venous and fall of arterial pressure. With tetanisation of the symp. they obtained (1) a brief rise of arterial and fall of venous pressure (20″ to 30″) followed by (2) a gradual return to and *beyond* the previous state of heightened venous lowered arterial pressure (2 to 3 min.). This hyperdilatation is note-

worthy; it is greatest after most intense stimulation, and is to be regarded as an exhaustion phenomenon—there remained after section a tonic influence—where? in the peripheral centres, say D. and M. With the plantar nerve, digital vein and digital artery, they obtained identical results. Hence the conclusion that there is no more reason to suppose dilatators in the sciatic than in the sympathetic, since in both the effects of irritation are the same, viz., primarily contraction; secondarily, dilatation. Resuming now the general question of dilatation, viz., paresis of contraction, whence may the suspension of activity proceed? from spinal or from peripheral centres? In the above experiments dilatation follows the interruption of an activity above the section, the exhaustion of an activity below the section; on the other hand nothing proves that the paralytic dilatation is a fatigue, for it should then always be preceded by a period of contraction, but we must admit that it is a primary effect. The effects cannot here be explained as an inhibitory interference of spinal over peripheral centres, and dilatation can probably occur in either of two ways, viz. by paresis of spinal centres or by paresis of peripheral centres.

Etude sur la Physiologie des Nerfs des Muscles Striés. S. TSCHIRIEW. (*Arch. de Phys.*, 1879, Nos. 3 and 4.—In a former paper (Tonus quergestreifter Muskeln, Arch. f. Anat. u. Phys., Phys. Abth. 1879, p. 89.)—T. pleads strongly in favour of the existence and reflex mechanism of muscular tonicity. The present paper continues to consider the theory of reflex muscular action by soliciting impression, and deals therefore with the nature, peripheral source, channels and presiding centre of the activity. Firstly, with regard to common sensibility, he remarks that normally muscle is insensible to pain by an instantaneous irritation, but sensitive to prolonged irritation, and also in certain pathological states (myalgia rheumatica, cramp, psoïtis, hysterical muscular hyperæsthesia, amyotrophic lateral sclerosis), and that the phenomenon of pain necessarily entails centripetal nerve-fibres. How do we judge of weight, or of the attitude and movement of our limbs? By some a specific sense of muscular tension ("the muscular sense") is imagined to arise in the contracting fibre; by others the sense of effort is attributed to the motor cell, being the subjective aspect and measure of its intensity of action; and by others still "muscular sense" is declared to be an entire misnomer, sensation being informed by altered tension, not of the muscle but of surrounding tissues. T. declares in favour of the peripheral

origin of a centripetal muscular sense by virtue of a special sensibility. Passing to the question of reflex movements by excitation of muscles, T. discusses the knee-phenomenon, and condemns the old theory of direct excitation of muscular fibre by mechanical tension; he refers it to a dragging upon the aponeurotic nerves by the percussion of the already stretched tendon, and asserts that the reflex is effected in that part of the cord whence the crural nerves originate—he estimates the interval between percussion and contraction at $\frac{1}{30}''$. His general conclusion is that muscles are connected with the cord by motor nerves and also by centripetal nerves, viz., by a nervous arc.

In a previous paper ('Sur les Terminaisons Nerveuses dans les Muscles striés,' Archives, 1879, p. 89) he claims to have proved that on striated fibres there are none but motor terminations, and that non-medullated nerves of muscle ending by arborisation in the aponeuroses are those of muscular sensibility.

Having regard to cases where there is "delay of pain," T. states the opinion that all sensations common and special are conveyed to the cord by common centripetal fibres, but are dissociated into separate channels by the agency of the cells of the grey matter. He anticipates, therefore, that delay of pain may be found associated with lesions limited to the posterior horns in correspondence with the affected district, and conversely that impairment of sensation without delay of pain should rule when lesions are confined to the posterior roots or columns.

T.'s paper concludes with a criticism of Krause's "hypothesis of discharge," a hypothesis based on a fallacious comparison of the motor end-plate with the plate of the electric organ. His previous paper ('Sur les Terminaisons,' &c.) went to prove that the terminal arborisation of the axis-cylinder, is the essential element of an end-plate, and not the granular layer. He adheres to Du Bois-Reymond's "modified hypothesis," advancing experiments to show how the shape of the end-plate should favour the concentration of the excitor influence of a disturbed electric equilibrium.

Dr. Gowers (*Med. Chir. Trans.*, vol. lxii. p. 269) estimates the mean "patellar tendon reflex" interval at $\frac{1}{10}''$ (·09 to ·15), and remarks that this corresponds with the *à priori* time value assignable to the reflex act (viz., ·045″ for conduction, ·05″ for central action, ·01″ for latent muscular period). He calls attention to a small rise in his tracings, about ·05″ after percussion, and asks whether this be not the indication of a *direct* muscular contraction by the *direct* irritation of muscular tension, the subsequent wave

indicating the indirect contraction by the *reflex* excitation of tension. G.'s account of the knee-phenomenon would thus include both its leading theories, viz., that of direct irritation and that of reflex excitation. For the front-tap contraction (a name given by G. to the gastrocnemius contraction, following a sharp tap on the anterior leg muscles), he finds an interval of ·04 to ·05, and concludes that the contraction is one of *direct* irritation. The ankle-clonus is regarded by G. as due to the direct stimulation of a muscular irritability, constituted by the reflex effect of tension, its frequency being 6 to 8 per sec., while the rare knee-clonus is regarded as reflex in mechanism, its frequency being 2·5 per sec. He considers clonus as an instance of a rhythmic action effected by a continuous stimulation.

NOTE.—In the course of measurements, which will be detailed elsewhere, of the interval between percussion and contraction, I found for the knee phenomenon ·04″ as its mean value in normal subjects (Gowers, ·09″ to ·15″; Tschiriew, ·033″). For the front-tap interval I found ·035″, ·04″, and ·04″ in three cases where the knee interval was measured to be ·035″, ·04″, and ·045″ (Gowers, ·04″ to ·05″). In two other cases (paraplegia) I found for the ankle-clonus a spasm frequency of 8 and 9 per sec., and for the knee-clonus of 9 and 10 per sec.

I may remark that a knee clonus does not seem to be so rare as it is usually stated to be. *A priori* we should not expect a rectus-clonus to be more unusual than a gastrocnemius-clonus, and it is simply owing to the respective circumstances of the two joints, that the spasms can commonly be elicited single in the rectus, multiple in the gastrocnemius. In point of fact a knee-clonus may be elicited by appropriate measures even in normal subjects, which is to the pathological phenomenon what the foot-trepidation of a normal subject is to its exaggeration—ankle-clonus. I have found the knee-clonus in the ankle-clonus cases I have examined by a flexing jerk of the rigidly-extended limb (best in the rigidity of paraplegics, where there remained some degree of voluntary motility).

The identity in the time-intervals of the single spasms at the two joints argues them to be of identical mechanism; the identity in the spasm-frequencies argues these also to be of identical mechanism. By other considerations that cannot be entered upon here, I am led to the conclusion that *all* the spasms above referred to are reflex from the spinal cord.

A. WALLER.

Primary Athetosis. GNAUCK. (*Arch. f. Psych.*, ix. p. 300.)—A girl, aged 13, who had always been healthy and had no predisposition to nervous disease, was seized with smarting pains in the right face. In eight days, continuous involuntary movements of the right hand and foot were observed, and shortly afterwards slight drooping of the right side of the face. Treatment with bromide of potassium was followed by disappearance first of the facial pains and then of the involuntary movements, these last

being completely gone in three months. The drooping of the face became less noticeable, but did not disappear. She remained well about three months, when her old symptoms reappeared. She noticed now that in certain places her sense of touch was not as acute on the right as on the left side. The facial pains ceased after an attack of epistaxis, but the other symptoms persisted. In this condition she consulted the author, who noted movements in the fingers, hand, forearm, toes, foot, and leg of the right side. The movements were slow, rhythmical, unceasing, involuntary, and yet, as it were, purposive, and they ceased during sleep. There was facial hypokinesis on the right side. The cutaneous sensibility was somewhat diminished in the parts in which the movements occurred. But there was no loss of power in the right extremities; the electric irritability was unchanged; the reflex excitability, including the phenomenon of tendon-reflex, was normal, and the circumference of the limbs was the same on both sides. Bromide of potassium was given in increasing doses, and the continuous current, from the cervical and lumbar regions of the cord downwards to the affected muscles, was applied every other day for about ten minutes. In a few months the patient had completely recovered.

The important points in this case are (1) the fact that the disease occurred in an individual who had previously enjoyed good health; (2) the partial recovery and subsequent relapse; and (3) the ultimate complete recovery of the patient. The case is one of idiopathic or primary athetosis, as distinguished from secondary or symptomatic athetosis, in which we have a history of hemiplegia, epilepsy, brain atrophy, &c. Of the primary disease only five cases are on record, and Gnauck gives a comparative analysis of them. In the only instance in which an autopsy was made, a focus of softening was found in the 'corpus striatum and lenticular nucleus' of one side. In the case just reported there was probably an affection of the outer part of the left half of the pons. The author distinguishes between symptomatic athetosis and cases of athetoid movements: in the former, the symptoms very closely resemble those of primary athetosis, in the latter, they differ from these in some important respects.

W. J. Dodds, M.D., D.Sc.

A Case confirming Cerebral Localisation. Dr. Tamburini (*Rivista Sperimentale di Freniatria, Anno V. Fascicolo III.*) gives an account of an imbecile, 45 years old, bearing the honoured name of Paul Veronese. He was subject to epileptic fits, which were often

followed by delirium. During an attack of this kind he killed an idiot with a knife. The epilepsy dated from infancy, and was accompanied by atrophy and paralysis of the left arm and leg. He was four years and a half in the asylum at Reggio, where he died. The fits had become so frequent that they could not be counted. The attacks were always preceded by an aura; then followed clonic movements, which began in the paralysed and wasted left arm. Sometimes the convulsions were limited to the arm, and extended to the face and leg of left side; sometimes they involved the whole body. The face was less developed on the left side than on the right. In fact the whole left side was smaller than the right; still the leg was not so much paralysed as the arm. The man's intelligence was very deficient, the power of speech limited, and the command of the vocal apparatus imperfect. Words were pronounced with great effort and with repetition and stuttering.

The left hemisphere of the brain was found to be normal, it weighed 510 grammes, but the right hemisphere was much smaller, weighing only 260 grammes; the whole brain weighed 930. On the right side, the ascending parietal convolution (the posterior median of Ecker) was atrophied and indurated, especially at its external part. This atrophy and induration involved the neighbouring portion of the ascending frontal gyrus anterior median of Ecker and the lower parts of the second and third frontal gyri. Behind this circle of sclerosed matter there was a cavity about the size of a pigeon's egg, full of purulent matter, extending through the left temporal lobe and occupying the place of the island of Reil, all vestiges of which had disappeared. The right thalamus opticus, crus cerebri, and surface of the pons on the right side were also found diminished in size and hardened in texture. The spinal cord was found atrophied, especially on the cervico-dorsal region. The sclerosed part examined by microscope showed an increase in the connective tissues and in the gyri round the fissure of Sylvius; amyloid corpuscles and white cells were found around the vessels.

Tamburini remarks that the atrophy of the left hemisphere continued into the crus cerebri, the pons and anterior pyramid of the same side, and then passing into the half of the spinal cord taken into connection with the atrophy and paralysis of the left side of the body, furnishes a striking proof of the dependence of the trophic and motor functions, not only in the cord and pons, but also in the cerebral hemisphere as well as of the crossing of the nervous tract denied by Brown-Séquard.

The loss of power in the left arm and leg and in the face is explained by the injury to the external portions of the ascending parietal, the ascending frontal, and the lower part of the third frontal.

The dissection of the brain, writes the learned physiologist, signally confirms the criterion for diagnosis brought to light by Hughlings-Jackson and experimentally confirmed by us (Tamburini and Luciani) that the lesion of the motor centre of the cortex cerebri may be diagnosed from the muscles first affected by the epileptic attack. The injury to speech is explained by the destruction of the island of Reil, which, in this case be it noted, was on the right side of the brain.

In considering the atrophy and sclerosis of the right optic thalamus, Tamburini enumerates the views of some distinguished neurologists on the function of this large basal ganglion. Luys holds that it contains four special centres for the representation of smell, sight, hearing and general sensibility. Ferrier thinks it to be a ganglion of interruption or centre of convergence of the sensory tracts. Fournier, Carville, Duret and Crichton-Browne hold that it is a centre of general sensibility. Nothnagel found that destruction of the optic thalamus was followed by a loss of sensibility and also of voluntary motion. Meynert, on the other hand, holds that the sensory impressions coming from the periphery are transformed in the optic thalami into movements; they are thus the automatic centres of reflex unconscious motions. Schiff, Lussana and Lemoigne concluded from their experiments that the optic thalami are centres for the motions of the arm and hand of the opposite side. The fact that in this case the atrophy of the optic thalamus was a continuation of the affection of the motor gyri, induces Dr. Tamburini to think that the thalamus is a conductor of the motor fibres, especially of the upper limb.

Hardening of the hippocampus major was found in this case; analysing the observations of five pathologists, he shows that it was found in 60 epileptics out of 272 dissections. He is of opinion that this lesion is only found in old cases of epilepsy. According to Meynert, the cornu ammonis contains peculiar pyramidal cells, but the destruction of the hippocampus causes no loss of any motor powers, nor does its removal bring any modification in epileptic convulsions. Tamburini recalls the views of Ferrier, who places the seat of olfactory sensation in the subiculum of the hippocampus. For these reasons he concludes that the hardening of

this portion of brain has no causal influence on the production of epilepsy. He considers that the morbid process, so well and learnedly described, commenced in the cortex cerebri, and then descended through the basal ganglia to the spinal cord.

Caizergues' Case of Cerebral Localisation.—Dr. Caizergues, in a pamphlet of eleven pages, which first appeared in the 'Montpellier Médical,' describes a case of cerebral hæmorrhage, in which the new views as to the functions of the cortex cerebri and white matter of the centrum ovale are well illustrated. The man, 38 years of age, was admitted into the hospital at Montpellier with mitral contraction and insufficiency and contraction of the aorta. The arteries were believed to be atheromatous. Suffering from diarrhœa he rose from his bed, and returned exclaiming, "I cannot move my fist." Then a feeling of tingling, similar to what he had felt in the arm, appeared in the right leg, and ten minutes later the whole right side was paralysed. He was heard to say to the religieuse, "Ma mère," and these were the last intelligible words he was heard to speak, though he tried to make himself understood by signs. The interne ascertained the presence of hemianæsthesia nicely limited to the right side of the body. There was no trembling or convulsions. He died about three hours after the first attack. An effusion of blood was found in the left hemisphere, which stretched in an oblique direction from above downwards and from behind forwards. The blood had raised the convolutions of the motor zone without injuring their tissue, and had gained the surface at one point near the union of the middle and upper third of the ascending parietal (posterior median of Ecker). The mass of the clot, about the size of a small apple, extended obliquely from below the fissure of Rolando to the fissure of Sylvius. Neither the nucleus lenticularis nor the nucleus caudatus was touched, but the internal capsule which separates them was found destroyed at its posterior and external part. The optic thalamus had escaped injury.

Dr. Caizergues thinks that the blood escaped gradually from the giving way of several miliary aneurisms. He holds that the paralysis of the arm and then of the leg was due to the destruction of the conducting white fibres of the centrum ovale, and not to any lesion of the motor area of grey matter, which Dr. Caizergues regards as left uninjured, although the blood forced its way to the surface at one point. The hemianæsthesia is explained by the destruction of the internal capsule of the corpus striatum, and

the aphasia is explained by the rupture of the lower pediculo-frontal fibres.

WILLIAM W. IRELAND.

Lactic Acid as a Hypnotic.—Dr. Maragliano has recorded in the *Rivista Sperimentale di Freniatria* (*Anno V. Fascicolo III.*) the result of some experiments upon the hypnotic and sedative properties of lactic acid upon the insane. He mentions two theories upon the production of sleep. Some physiologists have advanced that under the influence of fatigue oxygen disappears from the blood, causing inactivity of the nervous centres and sleep, but the provision of oxygen being renewed, their diminished powers are restored, and the man awakes. Others hold that, though the quantity of oxygen in the blood is not less during sleep than during the waking state, it is quickly absorbed by those materials, such as creatine and lactic acid, which are the result of the disintegrating activity of the bodily organism, and which require to be oxidised ere they are eliminated. When this oxidisation has been accomplished, the brain can again make use of the oxygen in the blood and resume its proper functions.

According to the experiments of Ranke and Preyer, the artificial introduction of lactic acid and other products of retrograde metamorphosis are capable of producing weariness and sleep. With this theory in mind a number of German physicians used lactate of soda or lactic acid in the treatment of sleeplessness, but the results were somewhat disappointing. Doctors Maragliano and Sepilli tried these drugs in about a hundred cases of insanity. They found that if given (lactic acid in doses of 8 to 10 grammes and lactate of soda 12 to 15 grammes) three or four hours before bedtime, they were sufficient to subdue the insomnia of quiet melancholia; but they had little or no effect if given immediately before going to bed. In more decided cases of agitation and sleeplessness they were found very inferior to chloral and morphia, besides being too costly for ordinary use. They were also apt to produce nausea and vomiting.

W. W. IRELAND.

Relations of Brain, Mind, and Higher Nerve Function.—In an able address delivered at the beginning of the session of the Pathological and Clinical Society of Glasgow, Dr. Alexander Robertson discusses some of the pathological and physiological relations of brain, mind, and higher nerve function. He begins by

giving a short sketch of the simpler forms of nervous apparatus in the lower animals, then proceeds to consider at length the theory of localisation of brain function, and particularly as it relates to the cerebral convolutions, "how far it seems worthy of acceptance, and how far it seems problematical." An epitome of physiological, pathological, and anatomical evidence in support of the theory of localisation is then gone over, from which it may be gathered that the greatest amount and most important part of the evidence is gained from a study of pathology, and more especially from the consideration of such diseases as aphasia, paralysis, and Jacksonian epilepsy.

Next in importance is the physiological evidence, in which some of the results obtained by Ferrier and Hitzig by electrical stimulation are discussed. Dr. Robertson says that of course there is no doubt about the facts elicited by these observers. It is in the interpretation of these facts that the difficulty lies; that is, whether the purposive movements, and so forth, brought about by stimulation of the cortex, are simply due to conduction of electrical currents from the grey matter of the surface to the basal motor ganglia; or depend on mere conduction of electricity from the motor centre to the basal ganglia by means of special fibres for the particular movements induced; or are due to reflex action, the surface in the area of the apparent motor centres being centripetal or afferent to the corpus striatum; or again are due, as Brown-Séquard proposes, to inhibition; or, lastly, as Hitzig himself advocates, to "what may be called a sensory explanation" of the apparent motor centres.

The anatomical evidence referred to by Dr. Robertson does not call for special reference, but he is of opinion that on the whole it leans to the theory of localisation. Dr. Robertson goes on to say that in the light of cortical localisation we arrive at a more satisfactory explanation of many forms of paralysis, and are able to account better for the grouping of symptoms and general course of some other forms of nervous disease. Take, says he, the case of general paralysis of the insane. In this disorder psychical disturbance usually precedes defective articulation, but occasionally they begin simultaneously. Now the presence of motor centres in the superficial grey matter affords a ready solution of the difficulty, showing how there may be associated two kinds of symptoms apparently so different from each other, as delusion and defective articulation, but really so closely allied since the motor intuitions of language are so deeply concerned in the exercise of thought. And it is

worthy of note that the adhesion of the pia mater to the convolutions is most marked in the frontal and parietal regions, a fact which supports the theory of localisation so far as it applies to motor centres.

The hypothesis of the psychical and motor functions being in such close relation also goes far to account for the frequent manifestation of motor symptoms in other forms of insanity.

W. G. Thompson, M.D.

Traumatic Insanity.—Dr. Brower (*Chicago Medical Journal and Examiner*, December 1879) believes that the production of insanity by injury to the head probably depends less on the nature of the injury and its associated phenomena than on the inherent peculiarities of the individual. Fatal injury to the brain may result from sudden violence inflicted on the skull without injury to the cranial bones. Dr. Brower had under observation during the late rebellion "a case of gunshot-wound of the head, that in twenty days resulted in death, and post-mortem examination showed no injury to the cranial bones, but an abscess in the brain and associated meningitis and encephalitis. The case was one, doubtless, of contusion of the brain with solution of continuity of its constituent elements, and without injury to the bone, the skull undergoing change of form suddenly, and the cerebral substance offering less resistance than its bony covering, extravasation and laceration of brain-tissue ensued." This author believes that if the connection between the injury and a criminal act is complete, "the history will show a neurophatic diathesis—will show that after the injury cephalegia was frequent, that the sleep was insufficient in quantity, or else disturbed by dreams; that after a time change was noticed in the emotional and then in the intellectual part of the mind; in rare cases both may occur simultaneously."

Bullet-wound of the Brain.—Dr. E. F. Brodie records (*American Practitioner*, January 1880) an interesting case of this form of injury. A man, aged 22, received on August 11, 1879, a wound of the cranium from a pistol-ball. The ball, which weighed half-an-ounce, entered at the supra-orbital foramen, fractured the orbital roof and temporal fossa, passed beneath the integument to a point two inches and a fourth above the meatus auditorius externus and out, dividing the posterior branch of the temporal artery. Several spiculæ of bone were removed, and about half-an-ounce of

cerebral tissue issued from the point of entrance. The patient was semi-comatose for about five days. On the tenth day he, having been kept on low diet, became so excessively hungry that his friends imprudently allowed him to eat bread and bacon, smoke a cigar, "and enjoy a convivial round generally," during which his outraged system revolted. He had hæmorrhage from the posterior branch of the temporal artery to the extent of five quarts. The wounds suppurated, and four days later a scale of lead, weighing 28 grains, was removed from the roof of the orbit, and also several large spiculæ of bone. In six weeks the wounds closed, leaving no symptoms beyond slight impairment of memory and stiffening of the muscles of the jaw. Subsequent observation of this case would be interesting and instructive.

ROBERT LAWSON, M.B.